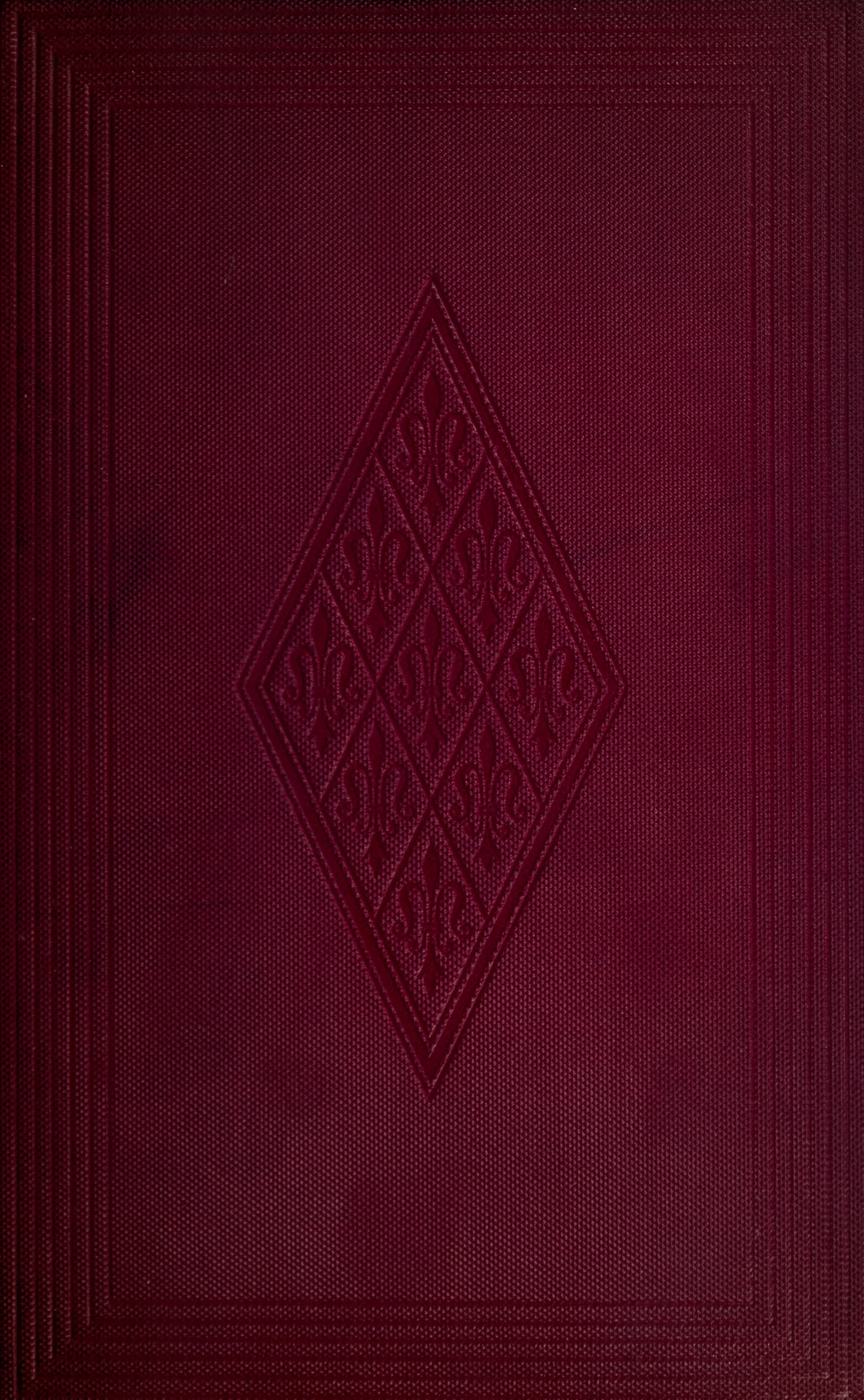

REPORTS

OF THE

SOCIETY FOR THE STUDY OF DISEASE IN CHILDREN.

VOLUME II.

SESSION OF 1901–1902.

EDITED BY

GEORGE CARPENTER, M.D.

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PUBLICATION COMMITTEE.

Appointed by the Council, July the 1st, 1902.

GEORGE CARPENTER, M.D.
EDMUND CAUTLEY, M.D.
THEODORE FISHER, M.D.
R. CLEMENT LUCAS, B.S.
SYDNEY STEPHENSON, C.M.

OFFICERS AND MEMBERS OF THE COUNCIL FOR THE SESSION OF 1902-1903.

ELECTED AT THE ANNUAL GENERAL MEETING,
JULY THE 16TH, 1902.

Council (1902-1903).

HENRY ASHBY, M.D. (Manchester).
FLETCHER BEACH, M.B.
HERBERT L. CARRE-SMITH.
EDMUND CAUTLEY, M.D.
WAYLAND C. CHAFFEY, M.D. (Brighton).
CLINTON T. DENT, M.C.
WALTER EDMUNDS, M.B.
LEONARD G. GUTHRIE, M.D.
ROBERT HUTCHISON, M.D.
FRANCIS JAFFREY.
T. SINCLAIR KIRK, B.CH. (Belfast).
DAVID B. LEES, M.D.
CHARLES J. MACALISTER, M.D. (Liverpool).
LEWIS MARSHALL, M.D. (Nottingham).
ROBERT G. McKERRON, M.D. (Aberdeen).
HENRY BETHAM ROBINSON, M.S.
A. ERNEST SANSOM, M.D.
GEORGE E. SHUTTLEWORTH, M.D. (Richmond, Surrey)
HAROLD J. STILES, C.M. (Edinburgh).
GEORGE A. SUTHERLAND, M.D.
JAMES TAYLOR, M.D.
ALFRED H. TUBBY, M.S.
RICHARD H. A. WHITELOCKE, C.M. (Oxford).
WILLIAM A. WILLS, M.D.

Honorary Treasurer.

R. CLEMENT LUCAS, B.S.

Honorary Secretaries.

SYDNEY STEPHENSON, C.M. } London.
GEORGE CARPENTER, M.D. } London.
THEODORE FISHER, M.D., Provincial.

RULES.

NAME.

1. The name of the Society shall be "The Society for the Study of Disease in Children."

OBJECT OF THE SOCIETY.

2. The object of the Society shall be the study of disease and of allied subjects in children.

CONSTITUTION.

3. The Society shall consist of Ordinary, Temporary, and Honorary Members.

QUALIFICATION FOR MEMBERSHIP.

4. All duly registered medical men shall be eligible for membership.

OFFICERS.

5. The officers of the Society shall be elected from the ordinary members, and shall include an Honorary Treasurer, Honorary Secretaries (one of whom shall be resident in the London postal district, and one in some other part of Great Britain or Ireland), and twenty-four other members, of whom at least one third shall not be resident in the London postal district. These together shall constitute the Council, and manage the affairs of the Society.

ORDINARY MEMBERS.

6. The names of candidates shall be proposed upon a printed form provided for the purpose, and signed by two or more members from personal knowledge. The names of candidates shall in the first instance be submitted to the Council, in which shall be vested the absolute power of declining to allow the

nomination to proceed. The proposal paper, after approval by the Council, shall be read at an Ordinary Meeting of the Society; and the ballot shall take place at the next meeting. No election shall be valid unless at least ten members vote, and no candidate shall be elected who fails to obtain four fifths of the votes recorded. No rejected candidate shall be eligible for proposal until twelve months have elapsed.

FORM OF ADMISSION.

7. Every Ordinary Member, after his election, and after payment of the annual subscription, and of the entrance fee (if any), shall sign the roll of members, declaring thereby his adhesion to the Rules of the Society.

TEMPORARY MEMBERS.

8. Any resident medical officer of a hospital or infirmary shall be eligible for election by the Council as a Temporary Member of the Society for a period of twelve months, without payment of any subscription or entrance fee. He shall be entitled to attend the ordinary Meetings, but he shall not have a vote, shall not be eligible for office, shall not be entitled to receive a copy of the 'Reports,' and shall not introduce visitors. If, at the termination of the period for which he has been elected a Temporary Member, he desires to become an Ordinary Member of the Society, he must go through the usual form of election, but shall be exempt from paying any entrance fee.

HONORARY MEMBERS.

9. The Council shall have power to propose for election as Honorary Members the names of persons eminent in medicine, surgery, or the collateral sciences. The Honorary Members, who shall not exceed twelve in number, shall be balloted for, after proposal by the Council, at a General Meeting of the Society, and be subject to the same rules as govern the election of ordinary members. They shall enjoy all the privileges of ordinary members, voting excepted. They shall not be called upon to pay any fee or subscription.

RESIGNATION AND EXPULSION OF MEMBERS.

10. Any member may retire from the Society on giving written notice of the intention to do so to one of the Secretaries and on paying any contributions that may be due.

The name of any member may be expunged from the Society's roll only at a General Meeting convened by the Council specially for consideration of the case. At least ten votes must be recorded, and four fifths of the votes, if recorded in favour of the expulsion, shall be decisive. Any person expelled in accordance with this rule shall forfeit all claim upon the Society.

SUBSCRIPTIONS.

11. The annual subscription for Ordinary Members shall be One Guinea, payable in advance at the beginning of the session in October. Ordinary Members elected after July the 31st, 1901, shall pay an entrance fee of One Guinea; but in the case of any member elected subsequently to Easter in any year the member shall not be called upon to pay a subscription during the ensuing session. Original Members pay no entrance fee. A member shall at any time be exempted from further annual subscriptions by paying a composition fee of Ten Guineas.

A member whose subscription is six months in arrear shall be reminded of the fact by one of the Honorary Secretaries; and if the amount due remain unpaid during the current year, his name may be removed by the Council from the list of members.

ELECTION OF OFFICERS.

12. The Officers of the Society shall be elected by ballot at the Annual Meeting. No member, except the Honorary Treasurer, may hold the same office for a longer period than three consecutive years. A list of names recommended by the Council for election shall be sent to each ordinary member at least fourteen days before the Annual Meeting of the Society. A name or names may be substituted on the voting paper for that of any member or members proposed by the Council for election. Two Scrutineers shall be appointed by the Chairman at the commencement of the Annual Meeting, and their duty shall be to receive and examine the balloting lists, and report the results to the meeting. In the case of equal voting the Chairman shall decide by lot.

HONORARY TREASURER.

13. The Honorary Treasurer shall have charge of the funds of the Society, and keep an account in an orderly and businesslike way of all sums that pass through his hands. He shall receive all moneys due to, and pay all moneys due from, the Society, only after the accounts have been passed by the Council and signed by two of the members present. He shall keep a printed receipt-book for subscriptions and fees, and every receipt shall be signed by him and countersigned by one of the Honorary Secretaries. He shall prepare an annual state-

ment of accounts, which shall be audited by two ordinary members of the Society not in office, and presented, with the Council's Report, to the next Annual Meeting. He shall keep a separate banking account in the name of the Society, and shall invest the funds from time to time as directed by the Council. The Honorary Treasurer shall be *ex officio* a member of Council.

HONORARY SECRETARIES.

14. The Honorary Secretaries (or one of them) shall conduct all correspondence, notify to new members their election, and convene, attend, and minute all meetings of the Society, of the Council, and of any committees appointed by the Council. They shall arrange the order of proceedings at all meetings. They shall submit to the Council all official communications and all nominations for membership in the Society. They shall keep a register of all cases and papers communicated to the Society, and, unless otherwise decided by the Council, shall act as editors of all publications authorised by the Council. They shall keep a roll of the officers and members of the Society. In the discharge of these various duties the Secretaries shall have power to employ clerical assistance, to use printed or typed forms, and to prepay postages and telegrams. They shall be *ex officio* members of Council.

MEETINGS.

15. These shall be *Ordinary*, *Special*, and *General*.

Ordinary Meetings shall be held at hospitals in London or elsewhere, or in places appointed by the Council. There shall not be more than eight Ordinary Meetings in the year, and of these one at least shall be held elsewhere than in London. The meetings shall take place upon the third Friday in the months of October, November, January, February, March, April, May, and June, at 5.30 p.m., unless otherwise arranged by the Council. They shall not be of longer duration than two hours, except by special resolution of the Meeting. Clinical and Pathological Demonstrations, and discussions thereon, shall take precedence over papers. Nothing relating to the laws or the management of the Society shall be discussed at an Ordinary Meeting.

Special Meetings may be convened by the Council for the purpose of discussing a subject or subjects deemed to be of particular interest in relation to disease in children, or to allied subjects.

A *General Meeting* shall be called by the Honorary Secretaries on the requisition of any five members of Council or of any twenty ordinary members of the Society. At least fourteen days' notice shall be given to every ordinary member of the

Society; and the character of the business to be transacted at such meeting shall be stated on the summons paper. No business other than that set forth upon the summons paper shall be considered at the meeting.

The *Annual General Meeting* shall be held at a time and in a place determined by the Council, fourteen days' notice having been given to every ordinary member of the Society. Its business shall include election of officers, presentation of the annual Report of the Council, and the discussion of any proposed alteration or alterations in the rules (notice of which shall be given in the summons convening the meeting). Ten ordinary members shall form a quorum. No change in the rules shall be valid unless three fourths of the votes are recorded in favour of the alteration. A notice, written or printed, may be served upon any member, either personally or by sending it through the post in a prepaid letter, addressed to such member at his registered address.

CHAIRMAN.

16. A Chairman, who must be an ordinary member of the Society, shall be chosen by show of hands or by ballot at the commencement of each meeting, whether ordinary, special, or general. He shall sign the minutes of the preceding meeting or meetings, regulate the proceedings, check irregularities, interpret the application of the laws, and enforce the recognised rules of debate. The Chairman shall have an ordinary and a casting vote.

PUBLICATIONS.

17. Reports shall be published in such manner and at such times as the Council may direct. A copy shall be forwarded free to every Honorary Member of the Society, and to every ordinary member whose subscription is not in arrear.

VISITORS.

18. An ordinary member may introduce two medical men as visitors at ordinary meetings of the Society. Visitors shall sign their names in the book provided for the purpose. On the invitation of the Chairman, visitors shall be permitted to take part in the discussions of the Society. The same visitor shall not be introduced more than twice during one session.

COUNCIL.

19. The Council shall have the management of the affairs of the Society, and shall meet as often as may be necessary for the

transaction of business. Seven days' notice of each meeting shall be sent by the Honorary Secretaries to every member of Council. Five members shall form a quorum. The Council shall determine questions, if necessary, by ballot. The Council, at its first meeting, shall elect from amongst its members a Chairman, to preside over its deliberations during the session. He shall not hold office for longer than two consecutive years. In his absence the chair shall be taken by some other member of Council, duly proposed and seconded. The Chairman of Council shall have a casting, in addition to an ordinary vote. The Council shall from time to time make regulations, issue instructions, appoint committees, and take any other lawful steps to further the good government of the Society. He shall prepare and present to the Annual Meeting a Report upon the state of the Society. The Council shall be empowered to fill all vacancies that may occur from death or otherwise between one Annual Meeting and another. It shall decide all questions relating to the publication of the 'Reports.' It shall annually depute three of its members, with the Honorary Secretaries, to form a *Committee of Reference*, the duty of which shall be to determine the acceptance of communications referred to it by the Honorary Secretaries. The Council shall be elected by ballot at the Annual Meeting. One third of its members, exclusive of the Honorary Treasurer and the Honorary Secretaries, shall be replaced annually by an equal number of ordinary members of the Society. An Extraordinary Meeting of Council may be called (1) by the Chairman of Council, or (2) by the Honorary Secretaries, and (3) shall be called by the Honorary Secretaries on the requisition of any five members of Council. Seven days' notice of such extraordinary meeting must be given in writing by the Honorary Secretaries to every member of Council upon a summons paper specifying the object for which the meeting is called.

ANNUAL REPORT OF THE COUNCIL.

Presented at the General Meeting on July the 16th, 1902.

THE Council takes pleasure in reporting that during the past Session the membership of the Society has increased from 237 to 290. They have to deplore the loss by death of one member, Dr. Alexander MacGregor, Physician to the North-London Hospital for Consumption and Diseases of the Chest.

The meetings of the Society continue to be well attended.

The provincial meeting was held on June the 21st, 1902, at the Children's Hospital, Pendlebury, Manchester, under the presidency of Dr. Henry Ashby. It was a distinct success.

The Council have decided to hold a Special Meeting in December, 1902, for the purpose of discussing *Tuberculous Peritonitis.*

They have further resolved that all the London meetings of the Society during the Session 1902–1903 shall be held at a central meeting place.

The Council have also decided that the next provincial meeting of the Society shall be held in June, 1903, at Brighton.

During the past session the Council have appointed a special committee to deal with and report upon the subject of *Precocious Puberty.*

The Hon. Treasurer's Balance-sheet shows that the financial position of the Society is in a satisfactory state.

Volume II of the Society's 'Reports' will be in the hands of members at the beginning of next Session. It will be edited by one of the Honorary Secretaries, Dr. George Carpenter, who was also responsible for Volume I. No change has been effected in the constitution of the Publication Committee.

TREASURER'S REPORT AND BALANCE-SHEET,

Made up to July the 16th, 1902.

Receipts.		£	s.	d.	Payments.	£	s.	d.
Brought forward from 1900–1901		166	14	9	Medical Society	8	8	0
Per Cash Book :					G. Pulman and Sons, Printing, etc.	20	12	3
Subscriptions, 1900–1901 ...	£68 5 0				Adlard and Son	126	1	8
„ 1901–1902 ...	173 5 0				Bale, Sons, and Danielsson	30	0	0
		241	10	0	S. Stephenson, Esq.	4	15	0
					Treasurer, Petty Cash	2	10	0
					Mr. H. Dickinson	15	2	0
						207	8	11
					Balance	200	15	10
		£408	4	9		£408	4	9

Of the above balance of £200 15*s.* 10*d.* the sum of £126 represents twelve composition fees.

We, the undersigned, have examined the foregoing accounts, together with the Vouchers and Bank Book, and find them to be correct.

R. CLEMENT LUCAS, *Hon. Treasurer.*

Auditors, { JAMES H. SEQUEIRA.
W. PAGE MAY.

Meetings of the Society were held during the Session of 1901–1902 *at the under-mentioned places:*

1. Friday, October the 18th, 1901.—Great Ormond Street Hospital for Children. Dr. D. B. LEES in the Chair.
2. Friday, November the 15th, 1901.—The Medical Society, Chandos Street, Cavendish Square, W. Mr. ROBERT JONES (Liverpool) in the Chair.
3. Friday, January the 17th, 1902.—The Evelina Hospital for Children. Southwark Bridge Road, S.E. Mr. A. H. TUBBY in the Chair.
4. Friday, February the 21st, 1902.—The Medical Society, Chandos Street, Cavendish Square, W. Dr. JAMES TAYLOR in the Chair.
5. Friday, March the 21st, 1902.—Paddington-Green Hospital for Children. Dr. HENRY ASHBY (Manchester) in the Chair.
6. Friday, April the 18th, 1902.—The Medical Society, Chandos Street, Cavendish Square, W. Dr. LEONARD GUTHRIE in the Chair.
7. Friday, May the 16th, 1902.—The North-Eastern Hospital for Children. Dr. WILLS in the Chair.
8. Saturday, June the 21st, 1902 (Provincial Meeting).—Pendlebury Hospital for Children, near Manchester. Dr. HENRY ASHBY (Manchester) in the Chair.
9. Wednesday, July the 16th, 1902 (Annual General Meeting).—The Hotel Cecil, Strand, W.C.; followed by a dinner. Dr. HENRY ASHBY (Manchester) in the Chair.

LIST OF MEMBERS OF THE SOCIETY.

OCTOBER, 1902.

(O.M. = Original Member. T. = Treasurer. S. = London Secretary. P.S. = Provincial Secretary. C. = Member of the Council. * = Life Member. C.C. = Chairman of Council.)

Elected

O.M. Abbott, Francis Charles, M.S., Assistant-Surgeon at St. Thomas's Hospital; Surgeon at The Evelina Hospital for Sick Children; 34, Weymouth Street, W.

O.M. Abrahams, Bertram Louis, M.B., Medical-Registrar at Westminster Hospital; 14, Welbeck Street, W.

O.M. Achard, Alexander Louis, M.D., 9, Blandford Street, Manchester Square, W.

1902 Adams, Charles E., 227, Gipsy Road, S.E.

1901 Allingham, Herbert William, Surgeon to H.M. The King; Surgeon at The Great Northern Hospital; Assistant-Surgeon at St. George's Hospital; 25, Grosvenor Street, W.

O.M. Alliott, Alexander John, M.D., Surgeon at The Hip Hospital, Sevenoaks; The Vine, Sevenoaks.

1901 Andriezen, William Lloyd, M.D., 7, Apsley Terrace, Acton.

1901 Archdale, Mervyn A., M.B., County Asylum, Rainhill, near Liverpool.

1902 Armour, Donald John, M.B., Bentinck Street Mansions, W.

Elected

O.M. Armstrong, Henry George, Medical-Officer at Wellington College; Wellington College, Berks. (C. 1900—1901 and 1901—1902.)

1901 Armstrong, John T., 109, North-End Road, West Kensington, W.

O.M. Ashby, Henry, M.D., Physician at The Manchester General Hospital for Children; Lecturer on Diseases of Children at Owens College; 13, St. John Street, Manchester. (C.)

O.M. Barnardo, Thomas John, 18, Stepney Causeway, London, E.

1900 Barr, James, M.D., Physician at The Royal Infirmary, Liverpool; 72, Rodney Street, Liverpool.

1902 Barrett, Alfred Keppel, 2, Peel Place, London, W.

1901 Battams, J. Scott, 123, Lavender Hill, S.W.

O.M. Beach, Fletcher, M.B., Physician at The West-End Hospital for Diseases of the Nervous System; Winchester House, Kingston Hill, Surrey. (C.)

1901 Beddard, Arthur Philip, M.D., Senior Assistant-Physician and Pathologist at The West-London Hospital; 44, Seymour Street, W.

1901 Benjafield, W. B., M.B., "Bletchenden," Lower Edmonton, N.

1900 Blagg, Arthur F., M.D., Physician at The Orthopædic Hospital for Children, Bristol; 28, Caledonia Place, Clifton, Bristol.

1902 Blaikie, James Brunton, M.B., Physician at The Hospital for Consumption, Margaret Street, W.; 22, Grosvenor Street, W.

O.M. Bluck, Walter Cardy, M.B., Byford House, York Road, Southend-on-Sea.

O.M. Blumfeld, Joseph, M.D., Anæsthetist at St. George's Hospital; Sloane Terrace Mansions, S.W.

1900 Bolus, Harry Boulcott, M.B., "Haddon," Beckenham, Kent.

O.M. Boyd-Wallis, Albert W., Brentwood, Essex.

O.M. Brightman, Frank, Aspley House, Broadstairs, Kent.

1901 Brown, John Henry, M.D., 14, Burngreave Road, Sheffield.

O.M. Browne, Hablot J. M., M.B., Hoylake, Cheshire.

Elected

1901 Bryant, John Henry, M.D., Assistant-Physician at Guy's Hospital; 8, Mansfield Street, W.

1901 Burgess, William Milner, 43, Nicoll Road, Harlesden, N.W.

O.M. Burghard, Fred. François, M.S., Surgeon at King's College Hospital and at Paddington-Green Children's Hospital; 86, Harley Street, W.

1901 Burt, Albert Hamilton, 34, Montpelier Road, Brighton.

O.M. Burton, Charles George, 97, Chatswood Road, Clapton, N.E.

1902 Buzzard, E. Farquhar, M.B., 33, Harley Street, W.

1902 Bythell, W. J. S., "Woodcliffe," Prestwich, Manchester.

1901 Campbell, Robert, M.B., Senior Surgeon at The Belfast Hospital for Sick Children; 63, Great Victoria Street, Belfast.

1901 Carless, Albert, Surgeon at King's College Hospital; 10, Welbeck Street, W.

O.M. Carmichael, James, M.D., Consulting-Physician at The Royal Edinburgh Hospital for Sick Children; 22, Northumberland Street, Edinburgh. (C. 1900—1901 and 1901—1902.)

O.M. Carpenter, George, M.D., Physician at The Evelina Hospital for Sick Children; 12, Welbeck Street, W. (C. 1900—1901). (S.)

O.M. Carre-Smith, Herbert L., 74, Holland Park Avenue, Holland Park, W. (C.)

1900 Cathcart, George C., M.B., 35, Harley Street, W.

O.M. Cautley, Edmund, M.D., Physician at The Belgrave Hospital for Children; Assistant-Physician at The Metropolitan Hospital; 15, Upper Brook Street, W. (C.) (*)

1901 Cave, Edward John, M.D., Physician at The Royal United Hospital, Bath; 20, Circus, Bath.

O.M. Chaffey, Wayland C., M.D., Physician at The Royal Alexandra Hospital for Children, Brighton; 18, Palmeira Square, Hove, Sussex. (C.)

Elected

1901 Chaldecott, John Henry, Anæsthetist at St. John's Hospital for Diseases of the Skin, at The Metropolitan Throat, Nose, and Ear Hospital, and at The Italian Hospital; 41, Welbeck Street, W.

O.M. Chapman, Charles William, M.D., Physician at The Hospital for Diseases of the Heart, Soho Square; 21, Weymouth Street, W.

1901 Cheyne, William Watson, C.B., C.M., F.R.S., Surgeon at King's College Hospital, and at Paddington-Green Children's Hospital; 75, Harley Street, W. (C. 1901—1902.)

O.M. Chubb, William L., M.D., Darenth House, Sandgate.

1901 Clapham, E. W., 72, Bishop's Road, W.

O.M. Clarke, Ernest, M.D., Surgeon at The Central London Ophthalmic Hospital; Ophthalmic-Surgeon at The Miller Hospital, Greenwich; 3, Chandos Street, Cavendish Square, W.

O.M. Clarke, James Jackson, M.B., Surgeon to Out-patients at The City Orthopædic and at The North-West-London Hospitals; 18, Portland Place, W.

1902 Cockburn, Robert P., 64, The Avenue, Ealing, W.

1902 Collie, Robert John, M.D., Medical-Examiner for The School Board for London; 25, Porchester Terrace, Hyde Park, W.

1902 Collier, H. Stansfield, Surgeon to Out-patients at St. Mary's Hospital, and Assistant-Surgeon at The Hospital for Sick Children, Great Ormond Street; 35, Westbourne Terrace, W.

1901 Collier, Mark Purcell Mayo, M.S., Senior Assistant-Surgeon at The North-West-London Hospital; Consulting-Surgeon at The National Hospital for Diseases of the Nervous System; 133, Harley Street, W.

1901 Coombe, Charles F., Surgeon at The Children's Hospital, Sheffield; 459, Crookes Moor Road, Sheffield.

1901 Cooper, Percy Roberts, M.B, "Glenthorne," The Downs, Bowden, Cheshire.

1901 Corner, Harry, M.D., Brook House, Southgate, N.

1901 Cox, Joshua J., M.D., 38, Deansgate, Manchester.

Elected

1902 **Cross, Ernest, W.**, "The Limes," Wallwood Park, Leytonstone.

1901 **Crossley, Leonard**, M.B., "Beechwood," Arnside, Carnforth, Lancashire.

1902 **Crowley, Ralph H.**, M.D., Physician at The Bradford Royal Infirmary; 116, Manningham Lane, Bradford.

1902 **Curtis, Henry J.**, B.S., Assistant-Surgeon at The Royal Hospital for Women and Children; 15, Harley Street, W.

1901 **Dawnay, Archibald Hugh Payan**, Anæsthetist at The Royal Orthopædic Hospital; 48, St. Mary Abbots Terrace, Kensington, W.

O.M. **Deas, Frank**, Thorndale, Merton Park, S.W.

O.M. **Dent, Clinton T.**, M.C., Surgeon at St. George's Hospital and at The Belgrave Hospital for Children; 61, Brook Street, W. (C.)

1901 **Dixon, R. Halstead**, M.B., 3, Florence Terrace, Ealing Common, W.

O.M. **Dockrell, Morgan**, M.D., Physician at St. John's Hospital for Diseases of the Skin; 9, Cavendish Square, W.

1901 **Dodd, Arthur Herbert**, 49, Church Road, Hove, Sussex.

1901 **Douglas, Arch. R.**, Royal Albert Asylum, Lancaster.

O.M. **Downes, Arthur**, M.D., Local Government Board Inspector; 46, Gordon Square, W.C. (C. 1900-1901.)

1901 **Drake-Brockman, E. F.**, Lieutenant-Colonel I.M.S., late Professor of Physiology and Diseases of the Eye at The Medical College, Madras, and late Superintendent at The Eye Infirmary, Madras, India; 14, Welbeck Street, W.

O.M. **Drew, Douglas**, B.S., Surgeon at The North-Eastern Hospital for Children, and at The Hospital for Women, Soho Square; 49, Devonshire Street, W.

1901 **Dun, Robert Craig**, M.B., Surgeon at The Liverpool Infirmary for Children; 6, Rodney Street, Liverpool.

1901 **Dunlop, James**, Aveley, Purfleet, Essex.

Elected

1901 Dunn, W. J. Robertson, 46, Wellington Terrace, West-Derby Road, Liverpool.

1901 Edelsten, Ernest Alfred, M.B., 370, Brixton Road, S.W.

O.M. Edmunds, Walter, M.C., Surgeon to Out-patients at The Evelina Hospital for Sick Children; Surgeon at The Tottenham Hospital; 2, Devonshire Place, W. (C.)

O.M. Elliott, Christopher, M.D., Senior Physician at The Bristol Hospital for Women and Children; 88, Pembroke Road, Clifton, Bristol.

1901 Ellis, W. McDonogh, M.D., Assistant-Physician at The Hospital for Women, Bath; Cedar Lodge, Bath.

1901 Evans, J. Howell, M.B., 28, Davies Street, Berkeley Square, W.

1901 Evans, Thomas Hy., 95, Shaw Street, Liverpool.

O.M. Ewart, William, M.D., Senior Physician at St. George's Hospital and at The Belgrave Hospital for Children; 33, Curzon Street, Mayfair, W.

O.M. Fenwick, William Soltau, M.D., Senior Physician at The London Temperance Hospital and Physician at The Evelina Hospital for Sick Children; 8, Devonshire Street, W.

1901 Fisher, H., Wycliffe, "Rathescar," Greenbank Drive, Sefton Park, Liverpool.

O.M. Fisher, Theodore, M.D., Pathologist at The Bristol Royal Infirmary, and Physician to Out-patients at The Bristol Hospital for Sick Children; Harley Lodge, Clifton Down, Bristol. (C. 1900—1901.) (P.S.)

1902 Flower, George John William, "Hendford," Yeovil, Somerset.

1901 Forster, Henry John, 119, Victoria Road, Romford, Essex.

O.M. Fowler, James Kingston, M.D., Physician and Lecturer on the Practice of Medicine at Middlesex Hospital; 35, Clarges Street, Mayfair, W.

O.M. Fox, T. Colcott, M.B., Physician for Diseases of the Skin at Westminster Hospital, and at Paddington-Green Hospital for Children; 14, Harley Street, W.

1901 Francis, J. E., University College Hospital.

Elected

1901 **Freeland, Ernest Harding**, Surgeon at St. George's and St. James's General Dispensary; 133, Harley Street, W.

1901 **Frost, Edmund**, M.B., "Chesterfield," Chesterfield Road, Eastbourne. (*)

O.M. **Fuller, Andrew**, Local Government Board Inspector; Oatelands Nook, Weybridge, Surrey.

1901 **George, Alfred Walter**, M.D., Surrey House, Shoot-up Hill, N.W.

1900 **Gervis, F. Harcourt**, M.D., 1, Lyncroft Mansions, West Hampstead, N.W.

O.M. **Giffard, Henry Edward**, Denham House, Egham.

O.M. **Giles, Leonard Thomason**, 13, Valley Bridge Parade, Scarborough.

O.M. **Godson, Clement**, M.D., Consulting-Physician at The City-of-London Lying-in Hospital; 82, Brook Street, W.

1902 **Goodall, Edward W.**, M.D., Medical Superintendent at The Eastern Hospital, Homerton, N.E.; The Eastern Hospital, N.E.

1902 **Goode, Henry Norman**, M.B., 5, Vicarage Gardens, Kensington.

1901 **Gordon, H. Laing**, M.D., 1, Honor Oak Road, Forest Hill, S.E.

1901 **Grant, J. Dundas**, C.M., Surgeon at The Central London Throat and Ear Hospital; 18, Cavendish Square, W.

O.M. **Gray, Alexander C. E.**, M.D., 1, Elm Park Gardens, South Kensington, W.

1901 **Gullan, Archibald Gordon**, M.D., 5, Canning Street, Liverpool.

O.M. **Guthrie, Leonard George**, M.D., Physician at The Hospital for Epilepsy and Paralysis, Regent's Park; Assistant-Physician at The North-West-London Hospital; Senior Physician at The Paddington-Green Children's Hospital; 15, Upper Berkeley Street, W. (C.)

O.M. **Gwynne, Charles Nelson**, M.Ch., Surgeon at The Children's Hospital, Sheffield; Highfield House, Sheffield.

O.M. **Hawken, Giles Lockwood Lang**, Surgeon at St. John's College, Hurstpierpoint; Hurstpierpoint, Sussex.

Elected

1901 Hawthorne, Charles Oliver, M.D., Physician at The Central London Ophthalmic Hospital; Assistant-Physician at The Royal Hospital for Children and Women; 28, Weymouth Street, W.

1901 Hayes, Thomas C., M.D., Professor of Obstetric-Medicine and Diseases of Women at King's College Hospital; 17, Clarges Street, Mayfair, W.

1901 Heywood, Charles Christopher, M.B., Irlams-o'-th'-Height, Salford, near Manchester.

1902 Hinds, Frank, M.D., Honorary-Surgeon at The Infirmary, Worthing; 17, Tennyson Road, Worthing.

1901 Hobhouse, Edmund, M.D., Assistant-Physician at The Sussex County Hospital, Brighton; 36, Brunswick Place, Hove.

1901 Hopkinson, Albert, M.B., 2, Parsonage Road, Withington, Manchester.

1900 Houghton, Philip, Lindfield, Sussex.

1901 Hutchison, Robert, M.D., Assistant-Physician at The London Hospital, and at The Hospital for Sick Children, Great Ormond Street; 22, Queen Anne Street, W. (C.)

O.M. Hutton, David Mount, M.D., 75, Hoghton Street, Southport.

O.M. Hutton, Henry Richmond, M.B., Physician at The Manchester General Hospital for Children; 16, St. John Street, Manchester.

1901 Huxley, Henry, 39, Leinster Gardens, Hyde Park, W.

O.M. Jaffrey, Francis, Assistant-Surgeon at St. George's Hospital; Surgeon to Out-patients at The Belgrave Hospital for Children; 33, Nottingham Place, W. (C.)

1901 James, Charles Alfred, 49, Stamford Hill, N.

O.M. Jessop, Walter Hamilton Hylton, M.B., Ophthalmic-Surgeon at St. Bartholomew's Hospital; 73, Harley Street, W.

1901 Johnson, L. Capper, Lidgate Park Road, Roundhay, Leeds.

1902 Johnson, Raymond, Surgeon at University College Hospital and at The Victoria Hospital for Children; 11, Wimpole Street, W.

Elected

1901 **Jones, E. White**, C.M., Surgeon at The Longton Hospital; Chaplin House, Longton, Staffordshire.

O.M. **Jones, Robert**, Surgeon at The Royal Southern Hospital, Liverpool; 11, Nelson Street, Liverpool. (C. 1900—1901 and 1901—1902.)

1901 **Kapadia, S. A.**, M.D., 49, Longridge Road, South Kensington.

1902 **Kelson, William Hy.**, M.D., Assistant-Surgeon at The London Throat Hospital; 15, Old Burlington Street, W.

O.M. **Kendal, Herbert William**, Surgeon at The Hospital for Sick Children and Women, Bristol; 1, Burlington Road, Redland Park, Bristol.

1901 **Kirk, T. Sinclair**, B.Ch., Surgeon at The Belfast Children's Hospital; and Assistant-Surgeon at The Royal Hospital, Belfast; 19, Great Victoria Street, Belfast. (C.)

O.M. **Knapton, George**, Craven House, Moss Lane, Manchester.

O.M. **Lack, Harry Lambert**, M.D., Ear and Throat Surgeon at The Children's Hospital, Paddington-Green; Surgeon at The Throat Hospital, Golden Square; 48, Harley Street, W.

1901 **Lacy, Alexander Gairdner**, "Saumarez," Sunninghill, Ascot, Berks.

1901 **Lake, Richard**, Surgeon at The Royal Ear Hospital; 19, Harley Street, W.

O.M. **Lambert, Sydney H. A.**, M.B., Gothic House, Harrow-on-the-Hill.

1901 **Langdon-Down, Percival L.**, M.B., 47, Welbeck Street, W.

1900 **Langdon-Down, Reginald Langdon**, M.B., 47, Welbeck Street, W.

O.M. **Lankester, Alfred Owen**, 5, Upper Wimpole Street, W.

O.M. **Lawson, Arnold**, M.D., Assistant-Surgeon at The Royal London Ophthalmic Hospital; and Ophthalmic-Surgeon at The Children's Hospital, Paddington-Green; 12, Harley Street, W.

O.M. **Lee, Philip George**, Surgeon at The Women and Children's Hospital, Cork; 25, St. Patrick's Hill, Cork.

Elected

O.M. Lees, David Bridge, M.D., Physician at St. Mary's Hospital, and Senior Physician at The Hospital for Children, Great Ormond Street; 22, Weymouth Street, W. (C. 1900—1901.) (C.)

1901 Leslie, W. Murray, M.D., 74, Cadogan Place, Belgrave Square, S.W.

1901 Levy, Alfred Goodman, M.D., Physician at The Farringdon General Dispensary; 16, York Place, Portman Square, W.

O.M. Lewis, Percy G., M.D., Folkestone, Kent.

O.M. Lister, Thomas David, M.D., Registrar and Pathologist at The East-London Hospital for Children; 95, Wimpole Street, W.

1901 Livingstone, George R., 35, George Street, Dumfries, N.B.

O.M. Lucas, R. Clement, B.S., Senior Surgeon and Lecturer on Surgery at Guy's Hospital; Consulting-Surgeon at The Evelina Hospital for Sick Children; 50, Wimpole Street, W. (T.) (*)

O.M. McCaw, John, M.D., Physician at The Belfast Hospital for Children; 78, Dublin Road, Belfast. (C. 1900—1901 and 1901—1902.)

O.M. McKenzie, Dan, M.D., Holly Lodge, Leytonstone.

O.M. McKerron, Robert Gordon, M.D., Senior Physician at The Children's Hospital, Aberdeen; 1, Albyn Place, Aberdeen. (C.)

1901 McNab, Alexander Anderson, M.B., 15, Wesley Road, Armley, Leeds.

1900 Macalister, Charles John, M.D., Physician at The Liverpool Royal Southern Hospital; 35, Rodney Street, Liverpool. (C.)

1901 Mackintosh, John Stewart, St. Ive, Platt's Lane, Hampstead, N.W.

1901 Mackintosh, William Alexander, M.B., 3, Park Terrace, Stirling.

1901 Maitland, Alfred Derwent, 1, Gloucester Villas, Hanwell, W.

Elected

1901 Makins, George Henry, C.B., Surgeon at St. Thomas's Hospital; Consulting-Surgeon at The Evelina Hospital for Sick Children; 44, Brook Street, W.

O.M. Malcolm, John D., C.M., Surgeon at The Samaritan Free Hospital; 13, Portman Street, Portman Square, W.

O.M. Marshall, C. Devereux, Assistant-Surgeon at The Royal London Ophthalmic Hospital; and Ophthalmic-Surgeon at The Victoria Hospital for Children, Chelsea; 112, Harley Street, W.

O.M. Marshall, Lewis W., M.D., Surgeon at The Children's Hospital, Nottingham; 42, The Ropewalk, Nottingham. (C.)

1900 May, W. Page, M.D., 9, Manchester Square, W.

1902 Maynard, Edward Forster, M.D., 108, Lansdowne Place, Hove.

O.M. Milburn, Charles Henry, M.S., Surgeon at The Victoria Hospital for Children, Hull; 158, Anlaby Road, Hull.

1902 Miller, Charles Hewitt, The London Hospital, E.

1902 Milligan, Wyndham Anstruther, C.M., Anæsthetist at The Great Northern Central Hospital; 104, Bethune Road, N.

1901 Milne, Robert, M.D., 32, Bow Road, E.

O.M. Moline, Paul, F., M.B., Anæsthetist at The Victoria Hospital for Children, Chelsea; 42, Walton Street, Chelsea, S.W.

1901 Monsarrat, Keith Waldegrave, C.M., Assistant-Surgeon at The Children's Infirmary, Liverpool; 11, Rodney Street, Liverpool.

1901 Montgomery, W. P., B.S., Surgeon at The Children's Hospital, Manchester; 15, St. John Street, Manchester.

1901 Morgan, Cecil Augustus, M.B., 6, Somerset Street, Portman Square, W.

1902 Morgan, George, Senior Surgeon at The Children's Hospital, Brighton; 6, Pavilion Parade, Brighton.

1901 Morison, Alexander, M.D., Physician to Out-patients at The Great Northern Central Hospital, and at The Children's Hospital, Paddington-Green; 14, Upper Berkeley Street, W.

Elected

1901 Morton, John Leyden, M.D., 15, Greville Road, Kilburn Priory, N.W.

1901 Morton, Samuel Ernest, 72, Burngreave Road, Sheffield.

O.M. Mossop, Isaac, Surgeon at The Bradford Children's Hospital; Victor Lodge, Manningham, Bradford.

1902 Mummery, John Percy Lockhart, M.B., 10, Cavendish Place, W.

1901 Munro, Henry Acland, M.B., "Lulworth," Rushey Green, Catford, S.E.

1901 Murtz, Anton Peter, Clareville, 48, Queen's Road, Finsbury Park, N.

1901 Nabarro, David N., M.D., Pathologist at The Evelina Hospital for Sick Children; 6, Crown Office Row, Inner Temple, E.C.

1901 Nash, W. Gifford, Surgeon at The Bedford County Hospital; Clavering House, Bedford.

1901 Nisbet, J. Towse, M.D., 7, Croxteth Road, Liverpool.

1901 Niven, Robert, Bethnal House Asylum, Cambridge Road, N.E.

1901 Oldmeadow, Lloyd, J. H., M.D., 15, Half Moon Street, Piccadilly, W.

1901 Oliver, John W., M.D., Hackney Infirmary.

1901 Oppenheimer, Heinrich, M.D., 63, Finsbury Pavement, E.C.

1901 Palmer, Frederick S., M.D., 7, Gordon Place, Gordon Square, W.C.

1901 Pare, John William, C.M., 64, Brook Street, W.

O.M. Parkinson, J. Porter, M.D., Physician at The London Temperance Hospital, and Physician to Out-patients at The North-Eastern Hospital for Children; 57, Wimpole Street, W.

1901 Parry, Leonard Arthur, M.D., 61, Church Road, West Brighton.

1901 Paterson, Alexander Gordon, M.D., South Lodge, Ascot, Berks.

1901 Pedley, R. Denison, Dental-Surgeon at The Evelina Hospital for Sick Children, S.E.; 17, Railway Approach, London Bridge, S.E.

Elected

1901 **Pendlebury, Herbert S.**, B.C., Assistant-Surgeon at St. George's Hospital; 44, Brook Street, W.

O.M. **Penrose, Frank**, M.D., Physician at St. George's Hospital and at The Hospital for Children, Great Ormond Street; 84, Wimpole Street, W. (*)

1901 **Pernet, George**, Pathologist at The Hospital for Diseases of the Skin, Blackfriars; 77, Upper Gloucester Place, N.W.

O.M. **Peskett, A. W. Chalmers**, M.B., Medical-Officer at The Nottingham Children's Hospital; 15, The Ropewalk, Nottingham.

1901 **Poland, John**, Senior Surgeon at The Miller Hospital, Greenwich; Surgeon at The City Orthopædic Hospital; 2, Mansfield Street, W.

1901 **Potts, William A.**, M.D., 160, Hagley Road, Birmingham.

O.M. **Power, d'Arcy**, M.B., Assistant-Surgeon at St. Bartholomew's Hospital, and Surgeon at The Victoria Hospital for Children, Chelsea; 10a, Chandos Street, Cavendish Square, W. (C. 1900-1901.)

O.M. **Prangley, Henry J.**, Tudor House, Anerley, S.E. (*)

1901 **Pratt, Eldon**, M.D., 36, Windsor Place, Cardiff.

1901 **Pritchard, George Eric Campbell**, M.B., 14, Cromwell Place, South Kensington, S.W.

1901 **Quennell, Arthur**, Brentwood, Essex.

1902 **Randell, R. M. Henry**, M.D., "Goodwood," Coper's Cope Road, Beckenham.

1901 **Raw, Nathan**, M.D., Mill Road, Liverpool.

1901 **Ray, John Howson**, Ch.M., Assistant-Surgeon at The Salford Royal Hospital; Surgeon at The Children's Hospital, Pendlebury; 22, St. John Street, Manchester.

1901 **Rendle, Arthur Bowen**, M.B., 43, Albion Street, Hyde Park, W.

1901 **Reeves, Henry A.**, Surgeon at The Hospital for Women and at The Royal Orthopædic Hospital; 70a, Grosvenor Street, W.

1901 **Rhodes, John Milson**, M.D., Didsbury.

Elected

O.M. Roberts, David Lloyd, M.D., Consulting-Obstetric-Physician at The Manchester Royal Infirmary; 11, St. John Street, Manchester.

1901 Roberts, George A., Evelina Hospital for Sick Children, S.E.

O.M. Robinson, Henry Betham, M.S., Assistant-Surgeon at St. Thomas's Hospital, and Surgeon at The East-London Hospital for Children; 1, Upper Wimpole Street, W. (C.)

O.M. Rogers, Bertram M. H., M.D., Pathologist and Physician at The Hospital for Sick Children, Bristol; 11, York Place, Clifton, Bristol.

1902 Romer, Harry, M.B., 132, Marina, St. Leonard's-on-Sea..

1901 Rose, Samuel Frank, Assistant-Surgeon at The National Dental Hospital; Dental-Surgeon at The North-Eastern Hospital for Children; 59, Queen Anne Street, W.

O.M. Roth, Bernard, Orthopædic-Surgeon at The Royal Alexander Hospital for Children, Brighton; 38, Harley Street, W.

1901 Rowland, Penry W., M.B., Royal Sea-bathing Hospital, Margate.

1902 Rowlands, Robert Pugh, The College, Guy's Hospital.

1902 Roxburgh, Alex. Bryce, M.B., Surgeon at The North-Eastern Hospital for Children, Surgeon at The Poplar Hospital, and Assistant-Surgeon at The London Hospital; 7, Henrietta Street, Cavendish Square.

O.M. Rugg, George L., 106, Knight's Hill Road, West Norwood, S.E.

1901 Rumboll, Sydney, Cavendish House, Leeds.

O.M. Sansom, A. Ernest, M.D., Consulting-Physician at The London Hospital, and Consulting-Physician at The North-Eastern Hospital for Children; 84, Harley Street, W. (C.C. 1900—1901 and 1901—1902.) (C.) (*)

O.M. Sansom, Henry Arthur, M.D., 127, West End Lane, N.W.

1901 Schilling, George, 58, Crystal Palace Park Road, Sydenham, S.E.

Elected

O.M. SEQUEIRA, JAMES H., M.D., Physician at The North-Eastern Hospital for Children; 13, Welbeck Street, W.

1901 SHACKLETON, WILLIAM WEBB, M.B., Grove House, Bushey, Herts.

1901 SHENTON, EDW., W. H., 20A, Hanover Street, Hanover Square, W.

O.M. SHUTTLEWORTH, GEORGE E., M.D., Ancaster House, Richmond Hill, Surrey. (C.)

1901 SIMS, F. MANLEY, Physician to H.R.H. the Duke of Cambridge; 12, Hertford Street, W.

1901 SIMSON, HENRY J. FORBES, 3, Charles Street, Berkeley Square, W.

1901 SKELDING, HENRY, B.C., Surgeon at The Bedford County Hospital; St. Loyes, Bedford.

1901 SMITH, FREDERICK JOHN, M.D., Senior Assistant-Physician at The London Hospital; 138, Harley Street, W. (*)

1901 SMITH, GUY BELLINGHAM, B.S., Surgeon at The Surrey Dispensary; 21, St. Thomas's Street, S.E.

1901 SMITH, T. GILBART. M.D., Physician at The London Hospital; 68, Harley Street, W.

1901 SMITH, TRAVERS, R.M., M.B., 13, Kilburn Park Road, N.W. (*)

1901 SMYTH, SIDNEY B., M.B., Physician at The Hospital for Sick Children, Belfast; Ulsterville House, Lisburn Road, Belfast.

O.M. SNAPE, ERNEST, M.D., 41, Welbeck Street, W.

1901 SNELL, SIMEON, Ophthalmic-Surgeon at The Sheffield General Infirmary; 70, Hanover Street, Sheffield.

1901 SOPER, WILLIAM, 307, Clapham Road.

O.M. SPOKES, P. SIDNEY, Dental-Surgeon at University College Hospital; 4, Portland Place, W.

O.M. STAUNTON, MICHAEL CORNELIUS, M.D., Visiting-Surgeon at The Children's Hospital, Dublin; 22, North Frederick Street, Dublin.

1901 STAVELEY, WILLIAM H. C., 13, South Eaton Place, S.W.

Elected

O.M. Stephenson, Sydney, C.M., Ophthalmic-Surgeon at The Evelina and The North-Eastern Hospitals for Children; 33, Welbeck Street, W. (S.)

1901 Steward, Francis James, M.S., Assistant-Surgeon at The Hospital for Sick Children, Great Ormond Street; 24, St. Thomas's Street, S.E.

O.M. Stewart, Hugh M., M.D., Walton House, Lordship Lane, S.E.

O.M. Stiles, Harold J., C.M., Surgeon at The Royal Edinburgh Hospital for Sick Children; 8, St. Colme Street, Edinburgh. (C. 1900—1901.) (C.)

1900 Street, Alfred Francis, M.D., "Burghfield," St. Mildred's Road, Westgate-on-Sea.

O.M. Summerhill, Thomas Henry, Surgeon at The Alexandra Children's Hospital, Rhyl; "Bodfor," Rhyl, Flintshire.

O.M. Sunderland, Septimus P., M.D., Physician at The Royal Hospital for Children and Women; 11, Cavendish Place, W.

O.M. Sutherland, George A., M.D., Physician at The Paddington-Green Children's Hospital; Assistant-Physician at The North-West-London Hospital; 73, Wimpole Street, W. (C.)

1901 Swan, Russell H. J., M.B., 8, St. Thomas's Street, London Bridge, S.E.

1901 Swanton, James Hutchinson, M.D., 40, Harley Street, W.

1901 Targett, James H., M.S., Assistant-Obstetric-Surgeon at Guy's Hospital; 6, St. Thomas's Street, S.E.

1901 Taylor, Frederick, M.D., Senior Physician at Guy's Hospital; Consulting-Physician at The Evelina Hospital for Sick Children; 20, Wimpole Street, W. (C. 1901—1902.) (*)

O.M. Taylor, James, M.D., Physician at The National Hospital for Paralysed and Epileptic, Queen Square, and Physician at The North-Eastern Hospital for Children; 49, Welbeck Street, W. (C.)

O.M. Thomson, StClair, M.D., Physician at The Throat Hospital, Golden Square, and Surgeon at The Royal Ear Hospital; 28, Queen Anne Street, W.

Elected

1902 Thorne, Atwood, M.B., 10, Nottingham Place, W.

1901 Thorne, W. Bezly, M.D., 53, Upper Brook Street, W.

1901 Thornely, William, M.B., 60, Herne Hill, S.E.

1902 Tod, Hunter, M.B., 7, Wimpole Street, W.

1901 Trout, J. H., Ensor, 412, Monument Road, Birmingham.

O.M. Tubby, Alfred H., M.S., Surgeon at Westminster Hospital; Senior Surgeon at The Evelina Hospital for Sick Children; Surgeon at The National Orthopædic Hospital; 25, Weymouth Street, W. (C. 1900—1901, 1901—1902.) (C.C.)

1901 Turnbull, George Lindsay, M.D., 76, Ladbroke Grove, W.

1901 Turner, H. Stanley, 16, Oakley Square, S.W.

1902 Turton, James, "Hatherly," Preston Park, Brighton.

O.M. Vacher, Francis, Consulting-Surgeon at The Wirrell Hospital for Children; "Glenmore," Shrewsbury Road, Birkenhead.

O.M. Vickery, William H., Surgeon at The Children's Hospital, Newcastle-on-Tyne; 52, Elswick Road, Newcastle-on-Tyne. (C. 1900—1901.)

1902 Walker, J. W. Thomson, C.M., 8, Cavendish Place, W.

1901 Wallace, Joseph B., M.B., 39, Battersea Rise, S.W.

1901 Walsh, David, M.D., Physician at The Western Skin Hospital; 70a, Grosvenor Street, W.

1901 Waring, John Alfred, M.B., 21, Derby Road, Nottingham.

1901 Weber, F. Parkes, M.D., Physician at The German Hospital, Dalston; Assistant-Physician at The North-London Hospital for Consumption; 19, Harley Street, W. (*)

1901 Wellby, Stanley, M.B., 27, St. Julian's Farm Road, West Norwood.

1901 Whipham, Thomas Roland C., M.B., Medical-Registrar at St. George's Hospital; 11, Grosvenor Street, W.

1901 Whitelocke, Richard H. A., C.M., Surgeon at The Radcliffe Infirmary, Oxford; 6, Banbury Road, Oxford. (C.)

Elected

1901 Williams, Chisholm, Surgeon to Out-patients at The City Orthopædic Hospital; 20, Bedford Square, W.C.

O.M. Williams, Dawson, M.D., Physician at The East-London Hospital for Children; 2, Wyndham Place, Bryanston Square, W. (C. 1900—1901 and 1901—1902.) (*)

O.M. Williams, Edward Cecil, M.B., Physician to Out-patients at The Bristol Royal Hospital for Sick Children; 9, Whatly Road, Clifton, Bristol.

O.M. Willey, Charles H., C.M., Surgeon at The Sheffield Children's Hospital; Wostenholm Road, Sharrow, Sheffield. (P.S. 1900—1901.) (C. 1901—1902.)

O.M. Wilks, Joseph Henry, B.C., Surgeon at The Children's Hospital, Sheffield; "Fairfield," Sharrow, Sheffield.

1901 Wills, William Alfred, M.D., Assistant-Physician at Westminster Hospital; Senior Physician at The North-Eastern Hospital for Children; 29, Lower Seymour Street, W. (C.)

O.M. Wilson, Kemlo R. M., M.D., Hanwell, W.

1901 Woods, John Francis, M.D., Medical-Superintendent at The Hoxton House Asylum, N.; 29, Queen Anne Street, W.

1901 Wright, Douglas, 229, Camden Road, N.

⁂ *It is requested that any inaccuracy in the foregoing list may be notified to either of the London Honorary Secretaries.*

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REPORTS
OF
THE SOCIETY
FOR THE
STUDY OF DISEASE IN CHILDREN.

SESSION of 1901-2.

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1. RHEUMATIC HEART OF GREAT SIZE; RECOVERY FROM BRACHIAL AND JUGULAR THROMBOSIS; RECOVERY FROM ADVANCED DROPSY.

By D. B. LEES, M.D.

THIS boy, aged 9 years, was admitted into the Great Ormond Street Children's Hospital in February, 1900, suffering from rheumatism, a greatly dilated heart, and thrombosis of the brachial and jugular veins on the right side. The right hand and arm were swollen, and the face puffy. He improved under treatment, and was discharged. Subsequently he was admitted again, having developed general dropsy, which four-minim doses of tincture of digitalis every six hours failed to relieve, but which began to improve directly the dose was raised from four to eight minims. The dropsy completely disappeared, and he was again discharged. The heart is extremely large, extending three fingers' breadth to the right of the sternum in the fourth space, and five finger-breadths to the left of the nipple line. Apical presystolic and systolic murmurs can be heard. At present there is no dropsy, and there has been no recurrence of the thrombosis.

(*Exhibited October the* 18*th*, 1901.)

Discussion.

Dr. Sansom asked whether the ailment was distinct rheumatic fever, or a subdued rheumatic affection. He had under his care at the London Hospital two cases which could be put into contrast with each other. One had a very marked rheumatic history, and repeated attacks of rheumatic heart trouble, and had had pericarditis. The other had no pericarditis, there was no obvious rheumatic history, but yet he was convinced it was a rheumatic heart, though of a slow-going character. In the one case it eventuated in obstruction and stenosis, and in the other a very large heart, extending over a very long period. There was also present well-marked rheumatic anæmia, which was a great cause in cases of thrombosis. He would like to hear where Dr. Lees thought that thrombosis took place. In his Lettsomian Lectures many years ago he (Dr. Sansom) described an analogous case, and the question raised was whether it was near where the thoracic duct entered.

(**Dr. Lees**.—This is the right side.)

Dr. Sansom (continuing) said that fact settled the point. He believed enlarged hearts in children got much better than one was justified in expecting. In his case now under care, though there had been marked pericarditis and very decided insufficiency, there was very striking improvement. A valve in a child might be very much swollen, but there might be great improvement, though not complete restitution. The suspicion of a presystolic murmur in the present case appealed to him (Dr. Sansom), because he thought that in the long-lasting cases there was often some induration at the mitral orifice, resulting in a definite constriction of it. There was no reason to think otherwise than that it might become smooth and fairly dilated, therefore sufficient for the auricle to supply the ventricle. At the same time the curtains and cords might resume a portion of their function.

Dr. Theodore Fisher (Bristol) asked whether, in the cases published by Dr. Lees with Dr. Poynton, of thrombosis with rheumatic heart disease, cultures were taken of the thrombi, and if so whether the organisms of rheumatism were found. When that was done in the case of thrombi, micro-organisms were almost invariably found. He thought the prognosis depended on the way in which the rheumatism had interfered with the nutrition of the heart muscle. He had seen the case of a man aged 64, who lived a busy life, eventually dying of bronchitis, but yet at the *post-mortem* examination his mitral orifice would only just admit one finger. In such a case probably the nutrition of the heart muscle had not been impaired.

Dr. Poynton (introduced) said the case was of great interest to him because the thrombosis had occurred in the brachial vein, and yet the patient had recovered in such a remarkable way. While Pathologist at St. Mary's Hospital he had three cases, one after another, of severe rheumatic thrombosis. In one, which was under Dr. Cheadle, both external jugular veins, both innominates, the subclavian, and the upper part of the superior vena cava became filled with thrombi. In another case of Dr. Cheadle's the same thing happened. When thrombosis occurred in the neck the condition might closely resemble parotitis, for which it might be mistaken, especially as the neck was stiff, and the movements of the head from side to side very much impaired. Another point was œdema of the face. We had been taught that œdema of the face in morbus cordis was much more common in children than in adults. It was not uncommon to find a child with morbus cordis who it was thought had renal disease. One of the causes of this might be thrombosis of the jugular veins. French observers had done more work at that subject than English. Some authorities said it was due to the actual rheumatic process, and the balance of evidence he thought favoured that view. It was said that thrombosis in rheumatism was more common in the lower extremities, but in the six cases he had seen it had occurred in the jugular veins, the brachials, and the subclavians. The cultures which he made were negative. The earliest symptom was usually a sudden swelling, either of the arm or neck, and in two of the cases there was urgent dyspnœa, and no doubt a certain detachment of thrombi and embolism, for in one case the patient became pulseless and dyspnoic for some hours, and in the other he rapidly sank.

The Chairman (**Dr. D. B. Lees**), in reply, said that no culture was made.

2. ARSENICAL NEURITIS CAUSED BY LIQUOR ARSENICALIS.

By D. B. LEES, M.D.

The patient, a girl aged 8 years, was treated by a medical man with fifteen-minim doses of the British Pharmacopœia preparation of liquor arsenicalis three times daily.

The drug was continued for *five weeks*, although the girl was not brought for inspection.

On admission to the Great Ormond Street Children's Hospital there was complete paralysis of the anterior tibial muscles of both legs, with absent knee-jerks, also some weakness of the extensors of both wrists, and wasting of the thenar muscles on both sides. Pigmentation was marked on the abdomen, and present in both axillæ.

Massage and the constant current were perseveringly used for six months. Improvement was at first very slow, and seemed to be almost stationary for some weeks, but was more rapid during the last month of treatment in the hospital. There is now distinct voluntary power over the dorsal flexors of both ankles, and much improved nutrition of the muscles.

(*Exhibited October the* 18*th,* 1901.)

Discussion.

Dr. Sutherland said it seemed curious that the young patient should have been able to retain such large doses of the drug. Was there any record of trouble in that respect?

Dr. C. O. Hawthorne said he thought the present case should not be used as a general warning against the employment of large doses of arsenic in cases of chorea. He (Dr. Hawthorne) for several years had been in the habit of giving choreic patients large doses of arsenic for a week or ten days at a time, but had never seen a patient develop, in the slightest degree, any evidence of peripheral neuritis. It was, he was convinced, a very valuable method of treating chorea. The risk was small, and arsenic was not peculiar in having risks attached to it; therefore it would be undesirable that this treatment of chorea should be prejudiced by such a case. He always used the hydrochloric solution of arsenic, and administered it well diluted in a half-tumblerful of water, ordering the patient to sip this during meal-times.

Dr. Chaffey (Brighton) agreed that arsenic should be given with caution, especially in grave cases of chorea. He had one case which was very much worse under arsenical treatment, and nearly died from exhaustion. It was only by the use of hypnotics that sleep could be induced. He had seen one case of death from chorea, in which arsenic was given but did not bring benefit. In that case hypnotics were not used. In chorea it was of the utmost importance to procure rest for the child. He did not feel at all sure that in the majority of cases of chorea arsenic benefited, but it seemed to answer best in mild cases when combined with iron.

The Chairman (**Dr. D. B. Lees**) said he agreed with Dr. Chaffey that arsenic should not be employed in acute cases, for which he had found benefit from pushing antirheumatic treatment freely. If the observations of Dr. Poynton and Dr. Payne were true, which led to the suggestion that acute chorea was due to the presence of the specific organism in the pia mater and blood-vessels of the brain, it would render treatment by means of antirheumatic remedies at all events reasonable. In a recent number of the 'Clinical Journal' he had related a case in which a patient who was violently choreic soon became enormously improved by such treatment; and he had seen several such cases. In the more chronic cases of chorea arsenic in large doses caused very much benefit if only administered over a limited time. His experience was that a dose of ten or fifteen minims of Fowler's solution thrice daily was usually well borne by the child, but he always gave it with bismuth after food, its administration being suspended on the appearance of vomiting, but resumed after a short interval. Once he caused arsenical neuritis, but it was soon recovered from. His object in showing the case was not to find fault with the administration of arsenic for chorea, but to call attention to its abuse.

3. LARGE RHEUMATIC NODULES WITH THICKENING OF THE TIBIA SIMULATING PERIOSTITIS; DISAPPEARANCE UNDER SALICYLATE.

By D. B. LEES, M.D.

This boy, aged 9 years, was admitted on November the 1st, 1900, with very large rheumatic nodules. The plaster cast on the table was taken from one of these on the occiput. The nodules were numerous on the scalp, also on the elbows, knees, ankles, and knuckles. The right tibia was thickened generally, with slight redness and slight tenderness, suggesting periostitis; it was for this condition that he was brought to Mr. Collier's out-patient department. Other rheumatic symptoms were slight. There was a little effusion into both knee-joints, and slight dilatation of the left ventricle of the heart, but no murmur.

Salicylate of soda in ten-grain doses every three hours was given for twelve days, and continued six-hourly for three months. At the end of that time the nodules had nearly disappeared.

(*Exhibited October the* 18*th*, 1901.)

Discussion.

Dr. C. O. Hawthorne said the point of interest which appealed to him was the size of the nodules, and that opened up a very large question of doctrinal diagnosis. No one with experience of rheumatism in childhood disputed that the eruption of subcutaneous nodules was a most valuable feature in the diagnosis. Those who held that rheumatoid arthritis was not rheumatism reposed, at one time, very great confidence upon the subcutaneous nodule. Later it was observed—and he had recorded and figured such—that considerable-sized nodules were present in the subcutaneous tissue in cases of rheumatoid arthritis. Then the question arose whether this could be used as an argument in favour of the rheumatic nature of rheumatoid arthritis. Here was a boy about whom there was no question of diagnosis, and who had considerable subcutaneous fibrous nodules. Therefore the occurrence of nodules in this boy was an illustration of the impossibility of drawing a firm clinical line of distinction between phenomena which might be called rheumatic on the one hand, and those which were rheumatoid arthritic on the other.

4. A CASE OF DIFFUSE PSEUDO-LYMPHOMA TERMINATING IN PERNICIOUS ANÆMIA.

By W. C. CHAFFEY, M.D.

The occurrence of pernicious anæmia in infancy is a rare event, and as in the present case it is associated with rickets, and with lymphomatous enlargement of several of the abdominal organs, it makes it interesting from a pathological as well as a clinical point of view. For the following brief but valuable notes I am indebted to our house surgeon, Mr. Fairhall.

Winifred S—, aged 11 months, was admitted to the Children's Hospital, Brighton, on July the 29th, 1901, suffering from well-marked anæmia and a large spleen. There was a history of lienteric diarrhœa, but nothing of note in the family history.

August the 1st.—Since admission the bowels have acted freely, but there has been no diarrhœa. The child has obvious rickets of the head and thorax, and is decidedly anæmic; but the long bones are not deformed, and her nutrition is fairly good. There is no œdema of the extremities, and there are no petechiæ of the skin. The abdomen is rather protuberant. The spleen is much enlarged, reaching down to the level of the umbilicus, and to within two inches of its left margin. The blood-count is as follows:—erythrocytes, 2,530,000 per cmm.; leucocytes, no increase (? absolutely decreased); poikilocytes, numerous; hæmoglobin, 44 per cent. of the normal.

August the 10th.—The patient's temperature has been subnormal, but this morning it has run up to 103° F. There is dulness at the pulmonary bases, but no obvious sign otherwise of inflammatory process. The spleen is as before. The liver can be felt below the costal margin, reaching somewhat lower than on admission. An attempt was made to view the optic discs; so far as could be seen there were no changes, and no choroidal hæmorrhages.

August the 17th.—The patient during the past week has been getting gradually weaker and more anæmic, and has developed some œdema of the lower extremities. She died this morning, twenty-one days after admission. During this time she had various drugs administered by the mouth; *e.g.* arsenic and iron; ipecacuanha and ammonia mixture; and ironol, castor oil, and tannigen, with a view to counteracting any poisonous toxine that might be present in the alimentary canal.

Post-mortem examination.—The body was very exsanguine. There was no marked wasting, the subcutaneous fat being in sufficient amount, and the muscular tissues fairly well developed though pale.

The thorax was much beaded. The heart, lungs, and lymphatic glands in the chest presented nothing abnormal.

Abdominal cavity.—The liver was rather large, but otherwise apparently normal to the naked eye.

The spleen measured 4 inches by 2½ inches; except that it was enlarged, and somewhat firmer than normal, nothing of note was observed.

The kidneys were both paler than normal; the left one was enlarged to twice the ordinary size. It retained its usual shape, covered by its capsule, which stripped off easily. To the naked eye it looked much like a large white kidney, and on being cut open the cortex appeared to be uniformly swollen and soft, but not diffluent. It was very pale in most parts, and showed but little differentiation between the cortex and medulla. There were a few firmer nodular portions of a more buffy tint, and several recent hæmorrhages into its substance, especially near the surface.

The right kidney was rather smaller than usual, pale on section, but apparently not otherwise affected. There were some yellowish stains about the calyces (? urobilin).

The bladder, ureters, stomach, pancreas, intestines, peritoneum, and mesenteric glands were all examined and found to be normal. There were no signs of scurvy or rickets in the long bones of the extremities, but the ribs all showed well-marked beading. There was no softening of the medulla of any of the bones, but rather a dry, pale appearance was noticed. There was no tubercle anywhere. Portions of the liver, spleen, and kidneys, after being acidulated with hydrochloric acid, were submitted to the action of a solution of ferrocyanide of potassium. The Prussian blue reaction, said to be characteristic of pernicious anæmia, was obtained in all three, but it was most marked in the case of the spleen.

Microscopical sections of these organs, made by the Clinical Research Association, show very well a small-celled infiltration along the capillary vessels. This is very marked at the peripheries of the lobules of the liver, the cells of which appear to be pressed upon, but not otherwise changed. In the kidney the small-celled infiltration is widely but not uniformly distributed through the cortex. The glomeruli and tubules are broken up and pressed upon, but their epithelial lining does not appear to be otherwise altered. In addition to the cellular infiltration there is seen, near the periphery of the cortex, a very delicate fibrillar network enclosing the small round-cells in places, which seems to indicate a distinct new growth of a lymphomatous nature; but it is probable that these fibrils are caused by coagulated fibrin, or are the remains of the capillary blood-vessels, so that the term "pseudo-lymphoma" might be the most appropriate to apply to the condition of the left kidney. In the spleen there is, in addition, well-marked thickening of the capsule and stroma. The characters of the blood during life aroused my suspicion that pernicious anæmia was present as a complication, and this opinion was confirmed by the examination after death; but I was not prepared to find the diffused lymphomatous changes in the liver and kidneys, especially in the latter.

I suppose the case must be viewed as primarily rickets, with possibly congenital syphilis, resulting in failure of the hæmatopoietic organs, and the appearance of lymphoid tissue as nature's last resource in an attempt at blood formation. I do not think it is a case of primary pernicious anæmia, as in that event the heart would almost certainly have been in a state of fatty degeneration, and there would have been subcutaneous and other hæmorrhages. The case may possibly be viewed as one of anæmia pseudoleukæmica, but the child was not seen early enough to enable me to determine whether it was primarily of that nature.

By way of comparison I may mention two other cases of pernicious anæmia which have recently been under my care at the Children's Hospital, Brighton. One was an infant admitted for anæmia, which gradually became extreme; the blood was kindly examined by Mr. Hamilton Burt, and shown to be affected similarly to that now under discussion. The infant died. A well-marked fatty heart and some tuberculous lesions in the right lung were found at the autopsy, which did not give rise to any well-defined signs during life.

The other case was that of a boy aged 9 years, which was reported in the 'Brighton and Sussex Medico-Chirurgical Society's Transactions,' just published. He was admitted suffering from well-marked anæmia, and died suddenly from blood extravasation into the substance of a fatty heart. His organs gave the characteristic Prussian blue reaction after death.

Both of these cases got rapidly worse, in spite of treatment and careful nursing. Indeed, I am not aware of any line of treatment that is of permanent avail in such cases, where a fatty heart and grave changes in the shapes and sizes of the red blood-cells have ensued; but we must hope that, by detecting the disease in its earlier stages, some good may result from therapeutic measures directed to improving the nutrition and the hæmatopoietic functions.

(*Read October the 18th*, 1901.)

Discussion.

The Chairman (Dr. D. B. Lees) said the Society was indebted to Dr. Chaffey for the communication, which was an attempt to throw light on a group of cases on which a good deal more light was needed, viz. those of enlarged spleen in young children. Syphilis was certainly the cause of some of them, but of how many had yet to be ascertained.

5. A CASE OF BIRTH PALSY.

By A. E. GARROD, M.D.

(Introduced.)

The child was born at full term, and there was no history of maternal illness during the pregnancy. He was breast-fed for three months, and then with cow's milk diluted. He was apparently strong and healthy in infancy, his only illness being an attack of bronchitis at three months of age.

The mother says that on first trying to walk, at three and a half years old, he was unable to stand or go alone, and always tried to walk on his toes. For the last twelve months he has been unable to see in the dark or in artificial light, and has had to feel for things, while in the daytime his sight has not been very good. Neither syphilis nor neurotic trouble was elicited in the family history. His surroundings at home were good.

Present condition.—The boy is aged 5¼ years, and is certainly mentally deficient, with a vacant stare. He cannot be taught to use the bed-pan. He is not malicious or cunning. There is no defect in speech. The muscles generally are in good condition; the thighs are adducted and the ankles extended, as the child sits in bed. The gait is a mixture of spasticity and ataxy in character, and he walks on his toes by rapid short steps with a wide base, holding up his hands to balance himself. He experiences difficulty in stopping and in turning round. No ataxy or choreiform movements of the arms are noticed. The grasps are fairly strong and equal. The arms are not rigid. The deep reflexes are increased; the triceps and supinator jerks are brisk. Clonus can be obtained in the left leg.

Eyes.—Both optic discs are paler than normal, with well-marked scleral rings of white colour. Both arteries and veins are small. Coarse and fine pigmentary changes are present.

The chest and abdomen present nothing abnormal.

(*Exhibited October the* 18*th*, 1901.)

Discussion.

Mr. C. H. Fennell (introduced), who exhibited the case for Dr. Garrod, drew attention to the retinitis pigmentosa, which was of interest in view of Dr. Batten's suggestion that syphilis was a prominent feature in a large proportion of cases of the disease.

Dr. Poynton (introduced) mentioned that there was a case of cerebral diplegia under his care in the out-patient department at the Great Ormond Street Children's Hospital which was getting progressively worse, a point of some interest as to the causation.

Dr. C. O. Hawthorne said it would be of great interest to have details of the family history; more especially bearing in mind the suggestion that syphilis entered into the case, and also in consequence of the existence of retinitis pigmentosa. Was there any blood relationship between the parents?

Mr. Carre-Smith asked what was the history of the birth of the child; was the labour a prolonged one?

Mr. Pernet said he thought the relationship of syphilis to cases of spastic paraplegia was a matter of great importance. In the present case, apparently, the retinitis pigmentosa pointed to syphilis. De Amicis,* of Rio de Janeiro, had placed on record a case in which there was undoubted syphilis in both parents. His case was a boy aged 3 years. Three healthy boys had been born, all still living, before the father contracted syphilis. This was followed by three abortions at the sixth, eighth, and eighth months respectively. The mother was then treated with mercury and iodide, and this was continued during the whole of the fourth (post-syphilitic) pregnancy, resulting in the birth of the spastic paraplegic child aforementioned. In some cases the condition has been described as a family disease. In this connection it is interesting to refer to two cases of spastic paraplegia from Dr. George Carpenter's clinic, shown by Mr. Carre-Smith before the West London Medico-Chirurgical Society. The children were sisters aged sixteen and five and a half years respectively. Dr. Carpenter, he understood, thought they were probably syphilitic. The mother had had seven premature deliveries (all these children died) and three miscarriages.

Mr. C. H. Fennell (introduced), in reply, said the spasticity and ataxic conditions were only noticed when it began to try to walk, which it did not do until three and a half years old. The family history was unimportant, and the present child was the only one. No evidence of syphilitic taint could be obtained. The child had had no other illness, and there was no suggestion of any hereditary disease in the family. According to the mother's account the labour had been a natural and normal one. In reply to Mr. Clement Lucas, as to how the retinitis pigmentosa could be accounted for, he said it was possibly the effect of congenital syphilis, but of that he could glean nothing.

Postscript by Mr. C. H. Fennell (*November the 8th,* 1901). —I find that I misunderstood Mr. Lister, who examined the eyes, and that he regards the eye condition as one of secondary retinitis pigmentosa. The diagnosis, I presume, lends additional strength to the suggestion of a syphilitic origin for the paralysis; he regards the original condition to have been one of choroido-retinitis, probably specific in nature.

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* "Die Little 'sche Krankheit und die hereditäre Syphilis," 'Arch. f. Derm. u. Syph.,' 1898.

6. CONGENITAL MITRAL STENOSIS IN A CHILD AGED FIFTEEN MONTHS.

By THEODORE FISHER, M.D.

THE boy whose heart is now exhibited was brought to the Out-patient Department at the Hospital for Children, Bristol, in August, 1901. The child was then rather small and anæmic, but not cyanosed. It was unable to walk. It was said to have been very blue at birth, and that there had been great difficulty in getting warmth into him. Attacks of difficulty of breathing, in which the child was described as having "gone a grey colour," had been frequent after birth.

The cardiac impulse was in the fifth space, just outside the nipple line. A rather high-pitched but not loud systolic murmur was audible all over the cardiac area, but was not conducted far into the axilla, and was not heard behind. The second sound over the pulmonary area was described as "clicking" in character, but elsewhere it was rather dull. At the apex a triple sound was audible. A deficiency in the intra-ventricular septum was thought to be the condition most likely to be present. There was nothing to suggest pulmonary stenosis, and the murmur thought to be characteristic of a patent ductus arteriosus was absent. The pulse-rate was 140 to the minute. A week later the child was brought up much worse. It was admitted into the ward, and died after being in the hospital a fortnight.

The heart was removed by the house surgeon; but having been taken away below the ductus arteriosus I afterwards went down to the parents' house and removed the arch of the aorta. The ductus arteriosus proved to be patent, but the diameter of its lumen was not more than one twentieth of an inch. The absence of a prolonged humming murmur was probably due to the small size of the arterial duct.

The chief feature of interest in the heart lies in the stenosis of the mitral orifice, which is not more than one tenth of an inch across. Not only is it interesting on account of the rarity of congenital mitral stenosis, but in this case there appears to be evidence also of the changes in the mitral valve which may be produced by endocarditis. The stenosis is of double character. There is a perforated diaphragm, which is attached to the upper part of the auricular surface of the mitral segments, and below the diaphragm the mitral segments and the associated chordæ tendineæ are much thickened and contracted. The heart is much hypertrophied, and weighed nearly 4¼ ounces.

Questioning the mother failed to elicit any history of rheumatism, either during pregnancy or at any other time. None of her other children, four in number, had suffered from rheumatism. This child was the sixth. Four were living and well, but one had died of diphtheria.

The records of cases of mitral stenosis which I have succeeded in finding have not been numerous. Sansom mentions a case in a child aged 2 months; the mitral valve was thickened, and presented granulations along the edges of the cusps; and he refers also to a somewhat similar case recorded by Ayroles.* Carmichael has met with a case where, in a girl aged 3 years, extreme mitral stenosis was found. Two orifices, not more than one thirty-second of an inch in diameter, were present. The ductus arteriosus was patent in this case.† Another example is recorded by Cotton, in which granulations were present both on the mitral and upon the segments of the aortic valve.‡

As will be seen from the description of the case recorded

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* 'Med. Soc. Trans.,' vol. xiii, p. 147.

† 'Edin. Hosp. Reps.,' vol. ii, p. 298.

‡ 'Trans. Amer. Pediat. Soc.,' 1900, vol. xii, p. 142.

above, the stenosis differed in one important detail from ordinary cases of mitral stenosis due to endocarditis. How the perforated diaphragm was produced is not quite clear. So far as I am able to understand the changes which occur in the auriculo-ventricular valve segments during their development, I do not see how a diaphragm can arise at the bases of these segments. The processes of endocardium which arise, and later, to some extent, atrophy, are situated at the extremities of what afterwards become the segments of the mitral valve. On the other hand, it is also difficult to understand how endocarditis can have produced the diaphragm, since this membrane did not occupy the usual site of rheumatic vegetations. In the aortic valve rheumatic vegetations may occasionally be seen to form a band which unites the segments, but I hardly think a band produced in this way can have been present here. Although it is not clear how this membrane can have been produced, it seems probable that endocarditis led to some change in the relative positions of the portions of the segments which develop from the ventricular wall and from the endocardium, and it is the endocardial portion which has persisted as the diaphragm.

In this case the existence of mitral stenosis was not suspected during life. No presystolic murmur was heard, and the triple sound audible at the apex was not of the rhythm commonly present in mitral stenosis.

It may be mentioned also that the patency of the ductus arteriosus was not suspected. I listened carefully in the second and third left intercostal spaces for the undulating but continuous murmur present in some cases of patent ductus arteriosus, but could hear only the systolic murmur that was audible elsewhere over the cardiac area. The narrowness of the lumen of the patent duct probably was the explanation of the absence of this interesting and curious murmur, which, it may be remarked in passing, has resembled, in the two cases in which I have heard it, the mixed sounds of the engine-room of a screw steamer.

(*Exhibited October the* 18*th*, 1901.)

7. CONGENITAL AORTIC STENOSIS FROM A CHILD AGED FOUR MONTHS.

By THEODORE FISHER, M.D.

A FEMALE infant, aged 4½ months, was admitted into the Bristol Royal Infirmary in May, 1899, and died the same day. In addition to broncho-pneumonia, the cause of death, the heart was found to be affected. All the segments of the aortic valve were much thickened, and adherent to one another. Some thickening of the flaps of the mitral valve and of the chordæ tendineæ was also present, but if any mitral stenosis existed it was slight in degree. Marked fibrosis of the muscular pillars, especially of the posterior pillar, was present. The left ventricle was much hypertrophied.

On inquiry from the mother, the child was said to have been a small, but apparently healthy baby. It never had attacks of cyanosis. The interesting fact, however, was mentioned that three months before its birth the mother suffered from severe pain in the left knee, and for several days she could only get about the house with great difficulty.

Congenital disease of the aortic valve appears to be more common than similar disease of the mitral orifice. Dr. George Carpenter quotes Rauchfuss as having recorded twenty-four cases of congenital aortic stenosis, the majority of which appear to have been due to endocarditis.* Cases in which two segments of an aortic valve are adherent to one another, and apparently have been so since birth, are not very rare. Whether such a condition is the result of an endocarditis or is a malformation may not be obvious. It is a curious fact that when two aortic segments only are present, the valve is far more likely to become affected during adult life with chronic endocarditis than when it is possessed of the normal three segments. Dr. Parkes

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* 'Congenital Diseases of the Heart,' p. 44.

Weber* argues from this that in some of the cases of disease of the aortic valve occurring in adults, where two cusps are found adherent, the adhesion which is thought to be the consequence of endocarditis has, in reality, existed from fœtal life. This may be so; but I have met with specimens in the post-mortem room which have illustrated various stages in the development of such adhesion during adult life. An inflammatory nodule appears at the junction of the attachment of two segments. This swelling generally spreads more or less equally into the adjacent segments, and may unite them together by a mass of considerable size, which, with other nodular thickenings that may have subsequently appeared, sooner or later becomes calcareous. The disease is one of adult life—in one instance only have I seen the nodular swellings under the age of twenty,—yet it is possible that a similar inflammation may attack the valves of the fœtus. This would be especially probable if one could connect the valvular disease with syphilis; but of such an association it seems to me there is little if any evidence. While, however, other causes of fœtal endocarditis besides rheumatism may exist, in the case of congenital disease of the aortic valve recorded above, the thickening, although well marked, resembled that commonly seen in rheumatism, and the history given by the mother leaves little doubt that rheumatism was the cause.

(*Exhibited October the* 18*th*, 1901.)

Discussion.

The Chairman (**Dr. D. B. Lees**) said the specimens were of extreme interest to him, because he had believed that congenital mitral stenosis did not exist. Such cases must be excessively rare. He would be very glad to hear what the experience of other members of the Society was on the point; *i. e.* whether any one present had discovered that lesion, or had ever heard a definitely presystolic murmur in a baby of under a year old.

Dr. Sansom agreed that congenital mitral stenosis, so far as he knew, was a myth, but he meant as a congenital anomaly. But the question of mitral stenosis was not settled, because it was the usual experience of all, that where there were congenital

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* 'St. Bart.'s Hosp. Reports,' vol. xxxv, p. 147.

anomalies in the heart that heart was more prone to the ordinary changes found in rheumatism, viz. endocarditis. There ought to be a distinction in the mind between congenital anomaly and congenital disease. It would not be incorrect to call the present case congenital mitral stenosis, but was it a malformation, or was there any intra-uterine endocarditis? He was convinced that the latter could exist. Dr. Tunstall sent him, and recorded very ably, a case in which there was distinct endocarditis in an infant with so-called congenital mitral stenosis, and in whom the changes were of the rheumatic character, but the mother had never suffered from rheumatism. That point required much further elucidation. But endocarditis in intra-uterine life was so rare that it was desirable for every case of it to be recorded. It might go on to fusion of the segments. That, however, was not a congenital anomaly, but a congenital disease. He thought Dr. Lees would agree that it did not disprove the contention that the change in the valve might be the result not of congenital malformation, but of congenital disease. He suggested that Dr. Theodore Fisher should modify the name of his specimen by calling it congenital mitral stenosis, which did not pledge anybody. His own contention was that it was a congenital disease, which might be started by a congenital anomaly, but it was not a congenital stenosis.

Mr. W. G. Nash (Bedford) said that last year he had a child under his care who, at birth, was apparently well, but in the first three or four months began to suffer from breathlessness and palpitation. It then became dropsical, and died at the age of six months. The mitral orifice was found very much puckered and diminished in size; apparently it was a case of mitral stenosis. He proposed to exhibit the specimen before the Society. The child seemed to have had no acute illness of any kind, and its heart gradually failed. There was no rheumatism in the mother, but after her confinement she had acute inflammation of the lungs. He thought that was a septic condition probably induced by the defective drainage of the house, and that the child suffered from it also.

Dr. Sutherland said, in response to the Chairman's question, that he had had a case eighteen months old in which a presystolic murmur was heard.

Dr. Theodore Fisher (Bristol) said he thought Dr. Sansom's remarks were of great interest and value. He himself felt no doubt that most congenital malformations on the left side of the heart were the result of intra-uterine endocarditis. A recent case had been recorded in America of endocarditis of both aortic and mitral valves, producing some stenosis of the mitral. The case referred to by Mr. Nash was probably also produced by endocarditis. But in his own specimen there was a curious abnormality which he could not see was capable of explanation on the intra-uterine endocarditis assumption.

8. A CASE OF CONGENITAL DEFORMITY OF THE LEFT SHOULDER GIRDLE AND SPINE.

By H. S. COLLIER.

(Introduced.)

The infant shown, aged 3 months, was the subject of a deformity of the shoulder girdle. It was brought to the Out-patient Department of the Great Ormond Street Children's Hospital on the previous day, and there had not been time to secure a skiagram. He thought the deformity was similar to that in the cases published years ago by Mr. Willett, of which he showed illustrations in the 'Medico-Chirurgical Society's Transactions.' He (Mr. Collier) also exhibited a process of bone which was removed by him three years ago from a similar case. There appeared to be a bony bridge extending from the backs of several vertebræ to the middle third of the posterior border of the scapula. He was also able to recognise that the scapula was ill-shaped, and that its vertebral border was too short. The back of the child was arched on one side, with well-marked lateral curvature. In the four cases he had seen of that trouble those were marked features. In Mr. Willett's first case several dorsal vertebræ were deficient, and there was not the normal number of ribs on both sides. The neck was short, and the bony bridge from the scapula was fused with the posterior surface of the lamina and spine of the third dorsal vertebra. The connection with the scapula in that case was by cartilage, and between the bone and cartilage there was a sort of fibrous material. The fusion to the scapula in his case was by means of very solid bone.

(*Exhibited October the 18th, 1901.*)

9. A CASE OF DEFORMITY OF THE HIP-JOINTS, RESEMBLING CONGENITAL DISLOCATION.

By H. S. COLLIER.

(Introduced.)

Mr. Collier showed a female child with deformity of the hip-joints, which superficially resembled that due to congenital dislocation of the hips. At three weeks old the baby was very ill, and abscesses were opened in the immediate neighbourhood of the hip-joints. A skiagram showed very markedly the separation of the femoral head epiphysis on each side, and there was a false joint between the diaphysis and epiphysis. He asked whether any member had had any experience of dissection of the condition; if so, whether there was fibrous union between the epiphysis and diaphysis, or whether there was a cavity.

(*Exhibited October the* 18*th*, 1901.)

10. DOUBLE FACIAL PARALYSIS FROM MIDDLE-EAR DISEASE; OPERATION; COMPLETE RECOVERY.

By ROBERT HUTCHISON, M.D.

The patient was a boy, aged 2 years and 2 months. He had enjoyed good health up to five weeks before he came under observation, when he was said to have suffered from an attack of "mumps." Shortly after that the mother noticed a discharge from both ears, and inability to move the face.

Examination showed complete paralysis, of peripheral type, of both facial nerves, with double otorrhœa, but without mastoid swelling. The parotid glands were normal, but there was slight enlargement of the cervical glands along the sterno-mastoid muscles. There was no other abnormality. The case was regarded as one in which both facial nerves were involved by the middle-ear disease. Mr. Ballance cleared out both mastoids. The day after the operation some return of power in the facial muscles was observed, which rapidly went on to complete recovery. At the present time (six months after the operation) the face is quite normal, both ears are soundly healed, and the hearing is good. (*Exhibited October the* 18*th*, 1901.)

Discussion.

The Chairman (**Dr. D. B. Lees**) said double facial palsy was very rare in children as well as in adults. Single facial palsy from middle-ear disease, however, was tolerably common. The double disease in the adult was sometimes due to syphilis affecting the bone on both sides. He inquired whether the patient's hearing was normal.

Dr. Sansom suggested, as a diagnostic expedient, that instead of pinching the child to make it cry, the feet should be tickled with the object of making it laugh. That did not avail in the present case. The face muscles appeared normal.

Dr. Hutchison, in reply, said he had only the mother's history to go by. There was a swelling on both sides, but the middle-ear discharge was noticed after that. There was no enlarged gland when the child first came under observation.

11. CASES SHOWING MULTIPLE CONGENITAL ABNORMALITIES.

By ROBERT HUTCHISON, M.D.

1. A boy, aged 16 months, is the first case for examination, a full-time child, the fifth in the family, the two eldest being alive and healthy, whilst the two immediately preceding the patient died before their second year, of bronchitis. The mother had been in poor health during her pregnancy.

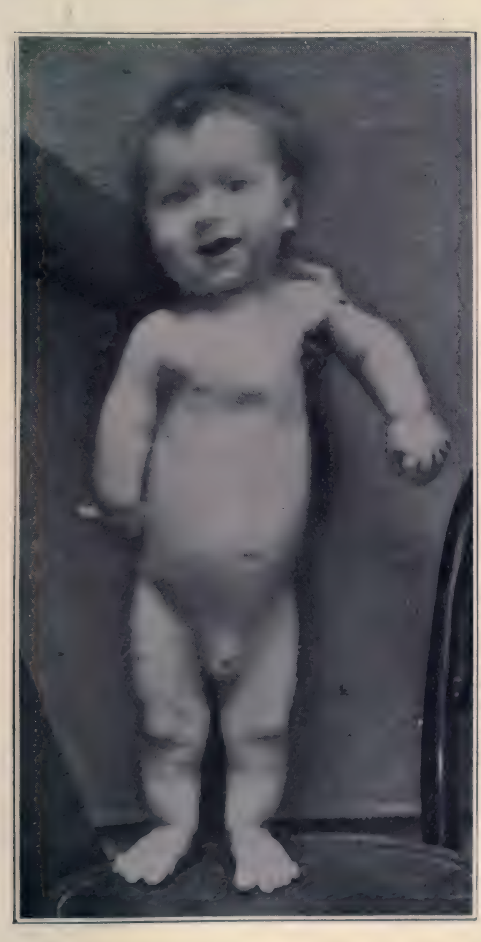
The child is well-nourished and well-developed, but exhibits a diffuse lipomatous nævus, involving chiefly the right arm and leg, which are fully half an inch thicker than the corresponding limbs. There is also a good deal of brownish pigmentation diffusely spread over both sides of the trunk and limbs, and extending beyond the limits of the nævus. In the right axilla there is a small fluctuating swelling, probably venous, and in the posterior axillary fold of that side, and over the fourth rib lower down, there are two small, apparently cystic, subcutaneous and movable swellings of doubtful nature. The child also exhibits (1) congenital adenoids of moderate amount; (2) a papillomatous growth in the centre of the dorsum of the posterior third of the tongue; and (3) a congenital cataract with shrinkage of the lens of the right eye. The other organs are normal.

2. A boy, aged 13 months, presents the following abnormalities :—(1) Congenital heart disease; (2) a rudimentary hare-lip; (3) six digits on each hand; (4) shortening of the long bones of the extremities; and (5) defective development of the gums.

The patient is the only child of healthy young parents. There is no history of congenital defects in either branch of the family. He was born at full time after an easy labour, and the mother's health during her pregnancy was very good.

The child is fairly well developed, weighs 14½ lbs., and is 26½ inches in height. His intelligence appears quite normal. The head is well formed, and in circumference measures 17½ inches. There is a rudimentary hare-lip on the right side (see Fig. 1). The upper gums are practically absent; the lower badly developed, and exhibit an isolated cartilaginous portion in the middle line in front, resembling a premaxilla. The mother states that this portion contained two teeth at birth. There are no teeth through anywhere, though there are indications of their being about to appear. The tongue and hard palate are normal, but the naso-pharynx appears to be narrow, and the child breathes through the mouth. All the limbs appear remarkably short in proportion to the trunk (see Fig. 1), and the finger tips when the child is standing up reach just below the level of the anterior superior spines.

Fig. 1.



The humeri are bent outwards about their middle, and each tibia presents a marked forward and outward bend in its lower third. The mother states that these bends were noticed at birth.

Measurements of the limbs.—Upper arm, circumference 5 inches; length from the acromion to the external condyle 3 inches. Forearm, circumference 5⅓ inches; length from the external condyle of the humerus to the end of the radius 3½ inches; total length of the arm from the acromion to the tip of the little finger 7¼ inches. Thigh, circumference 7½ inches; length from the anterior superior spine to the middle of the knee-joint 4¾ inches. Leg, circumference 6 inches; length to the external malleolus 3½ inches. Total length of the leg from the anterior superior spine to the outer border of the sole 9½ inches.

The hands are short and broad, and exhibit a supernumerary little finger on each. The fingers are all in one plane. The two middle fingers are of the same length, and on the left hand tend to exhibit a cleft between them. The feet are short, broad, and thick. There are no supernumerary toes. The nails, both of the fingers and toes, are extremely rudimentary.

A soft blowing systolic murmur can be heard all over the precordia, but the child exhibits no trace of cyanosis. The other organs, including the genitals, are apparently normal. (*Exhibited October the* 18*th*, 1901.)

Discussion.

Dr. Sutherland said that during the last fortnight he had seen a case almost identical with Dr. Hutchison's second patient. The child was three or four years old, with supernumerary digits, cardiac disease, and very much shortened limbs, also a hare-lip. It suggested achondroplasia. He proposed to show the case at the next meeting of the Society. The present case, he thought, seemed like one of achondroplasia; it had broadening of the toes and deviation from the middle line, which were regarded as practically diagnostic features. He did not remember what was the condition of the gums in his own case.

Dr. Hutchison, in reply, said he did not regard his case as one of achondroplasia, because he thought in that condition only the bones were involved, the soft parts being in folds merely because they were too large for the bones. Still, he was open to conviction on the point.

12. A CASE OF MULTIPLE CONGENITAL ABNORMALITIES.

By G. A. SUTHERLAND, M.D.,

AND

JACKSON CLARKE, M.B.

A boy, aged 2 years, presented the following abnormalities :—(1) congenital heart disease; (2) a rudimentary hare-lip; (3) six digits on each hand; (4) shortening of all the long bones of the extremities; and (5) defective development of the gums.

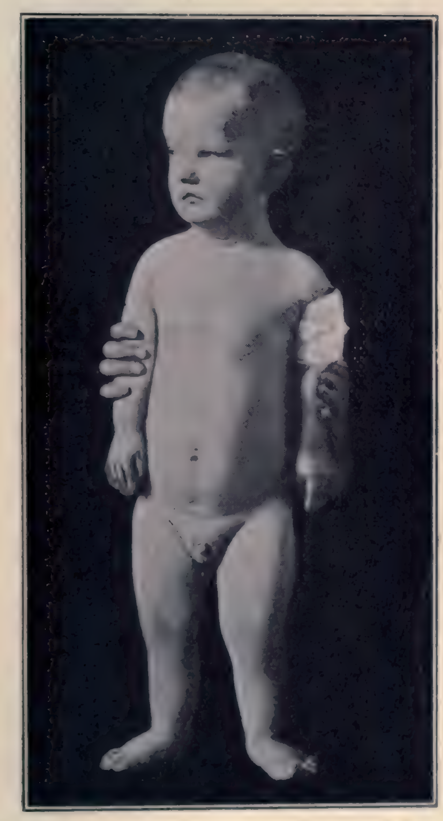
The patient was the son of healthy parents, who knew of no similar abnormalities in themselves or amongst their relatives. The second child, a boy, had also had six digits on each hand, a slight hare-lip, and blueness of the lips, nose, and ears; he died suddenly at the age of nine months from congestion of the lungs and heart disease. The first child, a girl, was now seven years old, healthy and well-developed in every way. The third child was the patient, and there had been no other pregnancies. The mother had noted, as regards the patient, that he had been blue and cold since birth. He had suffered from measles at the age of eight months, and had always been subject to bronchitis.

The conditions found on examination were as follows :—The boy was small for his age, and weighed 25 lbs. He was of a bright disposition, not at all passionate, always ready for play with his toys, and talkative, but his language was very difficult to follow. He could repeat simple words like "mamma," but the intellectual development was considered backward. The parietal and frontal eminences were well marked, but the occipital protuberance was rudimentary. The anterior fontanelle was almost closed. The circumference of the head was 18¼ inches. Over the scalp and the eyebrows the growth of the hair was very scanty, but the eyelashes were well developed. The palpebral fissures were small, the nose was "pug" shaped, and the upper lip sloped upwards from the sides, with a small notch in the centre. In addition to this rudimentary hare-lip, the lips were both drawn inwards, like those of an edentulous old person. The only explanation of this seemed to be imperfect development of the gums or jaws in the anterior portion, where the teeth were defective, there being only one incisor in the lower jaw, and none in the upper. The canine and molar teeth were present, and apparently normal. The palate was high and arched. The arms and legs were symmetrical, markedly short as compared with the length of the trunk, thickened, and somewhat shapeless as regards their outline.

Measurements of the trunk and limbs.—Trunk, from the coracoid process to the anterior superior spine, 11½ inches. Upper arm, circumference 6 inches; length from the acromion to the external condyle 4¼ inches. Forearm, circumference 5¾ inches; length from the external condyle to the end of the radius 4 inches. Whole upper extremity, from the coracoid process to the tip of the middle finger, 11½ inches. Thigh, circumference 10¼ inches; length from the anterior superior spine to the middle of the knee-joint 6 inches. Leg, circumference 7 inches; length from the middle of the knee-joint to the internal malleolus 5¼ inches. Whole lower extremity, from the anterior superior spine to the heel, 12 inches.

These measurements indicate that the shortening was proportionately greater in the upper arms and thighs than in the forearms and legs. A skiagram showed marked thickening in the upper ends of the humeri and the lower ends of the tibiæ. The hands also appeared to be broad, square, and shortened. There was a more or less complete sixth digit on each hand, attached to the little finger, and about half its length. Each consisted of a metacarpal and two phalangeal bones. The two middle fingers were of the same length, broad and thick, while the first and fourth digits were considerably shorter. When the hands were at rest, all the fingers tended to be spread out, but occupied the same plane at their junction with the hand. There was a marked anterior curvature in the femoral region, and an outward and backward curve in the tibial region, but no lordosis was present. He was able to stand and walk, but showed little confidence in himself, and usually dropped quickly into a sitting posture. The feet were short and broad and thick, with a tendency to pes cavus. The first and second toes were of the same length, and there were no extra digits in the feet.

Fig. 2.



Over the precordial area there were some dilated veins.

The heart was not enlarged, and the action was regular. Over the pulmonary area there was a blowing systolic murmur, well conducted along the sternum, and apparently congenital in origin. The nose, ears, chin, and extremities of the digits presented the dusky appearance of congenital cardiac disease. The penis was short, and the scrotum rudimentary in size. Both testicles were present in their normal positions.

We are unable to put this case into any class save that of congenital abnormalities. In some respects there is a marked resemblance to achondroplasia. It will be seen from the measurements and the figure, that the upper extremity, the trunk, and the lower extremity are of almost exactly the same length. The fact that Dr. Hutchison has presented a patient showing the same syndrome of abnormalities shows that a similar cause must have been at work in the development of both cases.*

(*Exhibited November the* 19*th*, 1901.)

Discussion.

Dr. Hutchison said the case bore an extraordinary resemblance to the one he himself showed at the Society's last meeting. Both had extra digits, a peculiarity of the gums, a congenitally abnormal heart, and a peculiar shortness of all the extremities. He was still unprepared to apply to it the term achondroplasia, as there was more in the case than there should be in that disease alone. If it were achondroplasia, one would not expect the soft parts to be so much shortened as the bones, and they would therefore hang down in folds. He saw his own case again on the previous day, and it was clear that the mental condition was good, so that could not be regarded as one of the essential features. He agreed to the term "achondroplasic," which did not commit one to very much.

Mr. Pernet pointed out that the hair on the patient's scalp was very scanty; there was also an absence of eyebrows, and the lower lashes were missing.

Dr. Shuttleworth (Richmond) said he would like to hear a little of the family history. Had there been supernumerary fingers in other members of the family? He thought the patient was mentally behind children of the same age, and the head was below the average size, only 18¼ inches, though he did not recognise any typically abnormal shape. One constantly saw developmental abnormalities associated with mental defects. He had at present under notice two cases of mentally deficient children each of whom had six toes.

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* *Vide* "Two Sisters showing Malformations of the Skull and other Congenital Abnormalities," by George Carpenter, M.D., vol. i of the 'Reports of the Society for the Study of Disease in Children,' pp. 110—118.

Dr. Sutherland, in reply, said that Dr. Hutchison and he were apparently agreed that the condition had not yet been defined fully. Marie had been lecturing on some cases of achondroplasia, and appeared to think that the condition was not a pure chondrodystrophy, but that the chondrodystrophy was the result of a general cause. He agreed that the hair of the patient was scanty, but to account for that one would probably suggest a myxœdematous tendency. He did not believe the child was a cretin. An infant brother of the patient shown, now deceased, had six digits on either hand, slight hare-lip, and blueness of the lips, nose, and ears.

13. HYDRONEPHROSIS IN A GIRL AGED FOURTEEN YEARS; IMPACTED CALCULUS AT THE LOWER END OF AN URETER SHOWN BY AN X-RAY EXAMINATION.

By DAVID WALSH, M.D.

Ellen G—, aged 14 years, came to hospital on November the 12th, complaining of rheumatic pains in her loins. There was a family history of rheumatic fever. The patient had always been healthy up to the present attack, which commenced about three months ago. For three weeks past she had attended a general hospital as an out-patient. The patient is well-nourished, but evidently in pain. The attack commenced suddenly three months ago. Pain was referred to both loins, but more particularly to the left. Movement of the body caused the patient much pain, and she had laced her stays to the utmost extent with an idea of obtaining relief. When quite still the pain sometimes ceased.

On examination there was dulness over the left flank and in the left lumbar region, where a tumour could be felt. Deep pressure caused pain in the region of the kidney. The appetite was good and the bowels regular; the lumbar spines were not tender to percussion; menstruation (which began at thirteen years old) occurred regularly every three weeks. The temperature was subnormal. The urine was loaded with urates and free from albumen, but showed a few hyaline casts. Mainly on these facts, hydronephrosis with an impacted calculus in the ureter was diagnosed.

An X-ray plate shows a large opaque band reaching from the kidney to the pelvis, and in the pelvis is a small stone. The value of the Röntgen method of diagnosis is well shown here. Many instances of stone impacted in the ureters have been published, but I know of none in which the hydronephrosis has been also recorded on the photographic plate. (*Exhibited November the 15th,* 1901.)

Discussion.

Mr. Clement Lucas suggested that if the stone were in the extreme lower part of the ureter there was another, and even more certain way of examining these cases than the author had described. It was an observation he made many years ago, that in cases of tuberculous disease of the kidney, where the ureter became thickened, that thickened ureter could invariably be found by examining *per vaginam.* He therefore suggested, before the present patient was operated upon, that an examination might be made in that way under an anæsthetic. Once it was clear where the stone was, there would be no difficulty in removing it. Probably it could best be removed through the bladder, by suprapubic cystotomy, aided, perhaps, by dilatation of the urethra; or if higher by laparotomy.

Sequel by Dr. David Walsh (*September the 5th,* 1902).—Subsequent operation showed no stone in the ureter. The value of this case lies in the demonstration of the extreme difficulty that is sometimes found in the interpretation of the facts presented in the X-ray plate, especially in dealing with small objects of little opacity buried in the deep tissue.

14. TUBERCULOSIS OF THE CONJUNCTIVA.

By SYDNEY STEPHENSON, C.M.

Emily H—, aged $1\frac{5}{12}$ years, was first seen as an out-patient at the North-Eastern Hospital for Children on October the 23rd, 1901.

History.—The child was weaned at thirteen months old. "A sort of lump" was noticed inside the right lower lid about two months ago, and the mother found that there was an ulceration inside corresponding to the swelling she had observed outside. The child "went off her food," and began to lose flesh about a month before the eye became bad. The patient is the younger of two children. There has been one miscarriage. The father is living. The mother (chest not examined) has a cough and sweats at night. The patient's maternal grandmother died of phthisis.

Present condition.—A thin, pallid, bad-tempered child, weighing $15\frac{3}{4}$ lbs. nude. The inguinal and the cervical glands are slightly prominent. The spleen is to be felt. Several small circular ulcers are present on the buttocks, three on the right, and one on the left side. The central depressed ulceration, which measures 2 mm. to 3 mm. in diameter, is surrounded by a red areola. The masses appear to be deposits of tubercle in the subcutaneous tissue. There are moist sounds over the back of the chest, ? broncho-pneumonic. Towards the centre of the free edge of the lower lid is a shallow notch, covered with greyish deposit. On everting the eyelid, a semicircular ulceration is seen to be continuous with the notch mentioned above. The base of the ulcer, which measures 7 mm. by 5 mm., is somewhat lardaceous and nodular. Several tiny, pale, translucent elevations, probably miliary tubercles, lie in the palpebral conjunctiva around the ulcer. There is no lacrymal or nasal tuberculosis. The right preauricular gland is somewhat swollen.

A diagnosis of tuberculosis of the conjunctiva was made, and the child was admitted into the hospital.

Scrapings from the conjunctival ulcer, after staining by the Ziehl-Gabbett and the Ziehl-Neelsen methods, were found to include a limited number of scattered tubercle bacilli. The organisms, as a rule, had to be carefully looked for, owing to their scanty numbers.

Progress.—When the child was seen two days ago (November the 13th), she was found to have developed double optic papillitis, some stiffness of the neck, and a typical tubercle in the choroid of her right eye. The tubercle lay not far from the optic disc, to its upper and inner side. It was crossed by a retinal artery. It was not altogether well defined, had a fawn colour, was more or less circular in outline, and was about half as big as the optic disc. The child's temperature was irregularly febrile.

Remarks.—Tuberculosis of the conjunctiva is one of the rarer affections of the eye. Indeed, this is the only case that I have recognised amongst about 3000 ophthalmic out-patients seen within the last few years at the North-Eastern Hospital. Its relative frequency has been estimated by Mules as 1 in 30,000, and by Eyre as 1 in 3125 eye patients. Young people below twenty years of age are far more liable to tuberculosis of the conjunctiva than older subjects, so that we should expect to meet with it more frequently in children's hospitals than elsewhere. Few cases have been reported in children as young as the present patient.

Conjunctival tuberculosis is essentially chronic, and must be especially distinguished from (*a*) Hunterian chancre, and (*b*) accidental vaccinia of the conjunctiva. In the present case chancre was excluded by the want of induration about the ulcer, and by the absence of secondary symptoms, although the ocular disease had lasted at least two months. Vaccinia of the conjunctiva is accompanied by symptoms of violent inflammation, and is an acute process. In favour of the tuberculous nature of the lesion were—1. The erosion of the border of the lower eyelid. 2. The existence of several small outlying tubercles in the conjunctiva. 3. The presence of "strumous nodules" in the buttocks. 4. The enlargement of the inguinal and cervical glands.* 5. The condition of the lungs. 6. A family predisposition, not well-marked, to tuberculous affections. It may be added that the appearance of the ulcer did not conform to that usually found in tubercle of the conjunctiva, namely, a nodulated ulceration surrounded by cockscomb-like granulations.

The positive diagnosis of conjunctival tuberculosis depends, of course, upon the discovery of the *Bacillus tuberculosis*. But in some of the reported cases—mainly, as it would seem, in those accompanied by prominent velvety granulations—there has been difficulty about this. Indeed, it is now generally advised to resort to the inoculation test, by introducing morsels of diseased tissue into the eye of a guinea-pig or rabbit (Villemin). In the present case, however, this was unnecessary, since there was not much difficulty in demonstrating the specific micro-organism in cover-glasses smeared with scrapings from the ulcerated surface. The bacilli, as stated before, were far from numerous. Thus conjunctival tuberculosis is no exception to the rule, *viz.*, that in chronic tuberculosis the microbes are few in number, and not always easy to find, even after differential staining, as by the Ziehl-Gabbet method.

The disease in the present case is clearly secondary, and forms a part merely of an acute miliary tuberculosis. Many of the cases of conjunctival tuberculosis, however, appear to be primary. It would scarcely be justifiable to operate upon the conjunctiva, seeing that the general illness is practically certain to have a speedy and fatal termination. Otherwise, I should destroy the ulcerated spot with the galvano-cautery, applied under a general anæsthetic, and afterwards use iodoform locally.

(*Exhibited November the 15th*, 1901.)

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* No particular stress can be laid upon the enlargement of the corresponding preauricular gland, because that is involved in primary syphilis, in vaccinia, and in many inflammatory conditions of the conjunctiva.

Discussion.

Mr. Pernet thought the condition appeared to answer to the miliary tuberculous ulcer which had been described by dermatologists, occurring sometimes about the margins of the lip. In that also the tubercle bacillus was not usually difficult to find.

Dr. C. O. Hawthorne asked if Mr. Stephenson would give his view as to the significance of the appearance of the fundus oculi in the case. There was a tubercle of the choroid and considerable swelling of the optic disc—several dioptres. Did Mr. Stephenson agree with the statement that a considerable swelling of the optic disc indicated a tumour rather than meningitis?

Mr. Sydney Stephenson, in reply to Dr. Hawthorne, said the summit of the discs could be seen with a + 9 D. lens, whereas the fundus generally could be seen with a + 2 D. lens, *i. e.* the discs were swollen 7 dioptres, or between 2 and 3 mm. The neuritis had distinctly the appearance of a tumour neuritis or choked disc. The swelling of the disc and the distension of the veins favoured that view, but he would not like to express a definite opinion in the present case whether it was due to tuberculous meningitis or to a tuberculous tumour. He thought it was a case of miliary tuberculosis of the meninges, because the child had tubercle elsewhere, probably in the spleen, as well as in the lungs and brain.

Postscript by Mr. Sydney Stephenson (September, 1902).—The child's temperature rose to 105·5° F., and she died two days after she was exhibited. Permission for a necropsy could not be obtained.

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15. A CASE OF HODGKIN'S DISEASE.

By C. O. HAWTHORNE, M.D.

Samuel N—, aged 6 years, was brought to the Royal Hospital for Children and Women on September the 2nd, 1901, on account of enlarged glands in his neck.

History.—He has always been a "trouble" to bring up. He had lumps in his neck at three years old, which disappeared. They returned last winter when he had pertussis, again disappeared, and returned a month ago after an attack of measles. He plays like other children, eats and sleeps well, his bowels are open daily, and he snores when he has a cold.

Family history.—His father died of consumption. His mother enjoys good health. There were nine children, three of the four now living have good health; two died of consumption and three in early infancy.

Examination.—His general nutrition is good, the face is well-coloured, but the lips and conjunctivæ suggest a moderate anæmia. The teeth are carious, and there are adenoids in the naso-pharynx. There is considerable enlargement of the lymphatic glands in the front of and under the upper part of each sterno-mastoid muscle. The posterior triangles are crowded with slightly enlarged, moderately hard glands. In the left axilla there are several glands moderately enlarged, and evidences of a similar condition in the right axilla. Everywhere the enlarged glands are non-adherent, readily isolated, and free from tenderness and other evidences of inflammation. *Chest:* there is some degree of pigeon-chest; at each base behind and in the lateral region numerous small moist râles are audible, with little or no impairment of the percussion note. *Abdomen:* the lower end of the spleen is appreciable on palpation; the liver is possibly slightly enlarged. The urine is free from albumen. An ophthalmoscopic examination revealed a normal fundus. The temperature for the most part has been normal or subnormal, but has occasionally reached 100° F. *Blood:* the red cells are 2,300,000 per c.mm.; the hæmoglobin is 70 per cent.; the proportion of white cells is approximately normal; a differential count of white cells by the Clinical Research Association yielded the following results:—polymorphonuclear neutrophiles, 46·2 per cent.; small lymphocytes, 43 per cent.; eosinophiles, 3·6 per cent.; large lymphocytes, 7·2 per cent. (*Exhibited November the 15th,* 1901.)

Discussion.

Dr. Sansom asked whether the boy's spleen was enlarged, and whether there was any marked anæmia, *i. e.* deficiency of blood-corpuscles or of hæmoglobin. The signs in the lungs were very peculiar in a disease of that kind. In a case he had carefully watched there had been strands of lymphoid or morbid tissue, and he thought it probable that there had occasionally been a congested condition of the neighbouring lung, without actual infiltration or breaking down. No doubt there had been hæmoptysis in the present case, and that symptom in the case he had in mind lasted a long time. The general story was that it had a general analogy to tubercle, but it was not like tubercle. It was very chronic in the sense that it lasted longer than one would expect; but Hodgkin was quite right in calling it a malignant disease.

Dr. Hutchison suggested that the case was not one of Hodgkin's disease, but of tuberculosis of the glands. He had been impressed with several cases of a similar sort which he had followed out, in which the diagnosis of Hodgkin's disease was proved to have been wrong. A boy, slightly older than the present patient, had glands in the axilla and neck, a large spleen, and a large proportion of leucocytes. Every one thought it was Hodgkin's disease, but, as no rapid change ensued, a surgeon cut out one of the axillary glands, and it was demonstrated to be tuberculous. The only safe criterion for establishing the diagnosis was staining for the bacillus; and some pathologists went so far as to say that the only proof was the inoculation of an animal with a caseous gland. He was convinced that the blood condition was of little assistance in the diagnosis. The last case of the kind he examined was a very acute one. Every one called it lympho-sarcoma; there were a large number of leucocytes—about 40,000, and the lymphocytes were predominating. Yet the glands were swarming with tubercle bacilli. He suggested that in the present case one of the glands should be excised, sections cut, and that they should be stained for the tubercle bacillus. The family history in the present case favoured the tuberculous view. The signs at the bases of the lungs could be explained on the assumption that the child had enlarged glands in the mediastinum, which probably interfered with the return of the venous blood.

Mr. Pernet said he was inclined to agree with Dr. Hutchison, to some extent, because the present patient had had measles, and the glands had become much larger since then. Measles often did start the tuberculous process, and he thought the glands might have become much larger from the measles liberating the bacilli into the circulation.

Mr. Clement Lucas said he could not admit that Hodgkin's disease was non-existent, and that all supposed cases of that nature were really tuberculous, as he understood the Americans were inclined to believe. Only a few days ago a patient in Guy's Hospital under his care died of Hodgkin's disease. He had been operated upon for the removal of his glands, and the disease developed afterwards in both armpits, in the groins, and on both sides of his neck, and he died of pneumonia. His medical colleague fully agreed with the diagnosis of Hodgkin's disease before death, and a *post-mortem* examination verified it.

A Member said he had had two cases of lymphadenoma this year, from both of which inoculations were made, but the guinea-pigs showed no signs of tuberculosis.

Dr. C. O. Hawthorne, in reply to Dr. Sansom, said the lower end of the spleen could just be felt, but the significance of that observation was discounted by the fact that the boy had some degree of pigeon-breast. Possibly, therefore, the spleen was felt not because it was enlarged, but because it was displaced downwards. The blood showed 70 per cent. of hæmoglobin, but there was no disturbance of the usual proportion of red to white corpuscles. The importance of the histological examination was that the proportions of different kinds of white corpuscles were disturbed in the direction of producing a great increase in the lymphocytes. The belief expressed by Dr. Hutchison, that there was no such condition as Hodgkin's disease, raised a very large and grave pathological and clinical question which he was not there to discuss. But he was willing to grant that if there were no such thing as Hodgkin's disease, then assuredly this was not an instance of it. But he held that if the usual clinical landmarks held good, it was a case of Hodgkin's disease. The character of the glandular enlargement was different from that of tuberculous adenitis. There was, for example, the non-adherence of the lymphatic glands to their neighbours, and the fact that the boy was active and fairly well in health. He appreciated the suggestion that one of the glands should be excised and examined, but he could only promise to consider it. He adhered to his original view.

16. A NOTE ON A FAMILY EPIDEMIC OF IMPETIGO CONTAGIOSA BULLOSA ORIGINATED BY PEDICULI CAPITIS.

By GEORGE PERNET.

Whilst in charge of the Skin Department of University College Hospital last September four children of the same family were brought to the out-patient room. I will shortly describe the cases in the order in which they were observed.

Case 1.—The patient was a boy, aged $2\frac{10}{12}$ years, with an eruption of two days' duration. It had commenced as a "water-blister" on the left forearm, and had then spread to other parts of the body, trunk, etc. The mother, an intelligent woman, stated that the lesions commenced as vesicles of the size of hemp-seeds which rapidly enlarged, attaining the diameter of a pea and later of a halfpenny, when they burst. As a result of this there were found on examination about a dozen recent superficially excoriated areas, about one inch in diameter, and looking very like what is observed in involuting pemphigus neonatorum. On the outer side of the left leg there was a vesicle of the size of a lentil, with clear contents and an erythematous areola. The mother said that the child had been feverish; on taking the temperature, however, it was found to be only 99·6° F.

Case 2.—The patient was a boy, aged 8 years. He had only been affected since the previous night, when a "water-blister" of the size of a pea was noticed on the back of the head. It was stated to have burst, and the lesion to have since extended. When I saw him there was a crusted, superficially excoriated area a quarter of an inch in diameter, with surrounding redness.

Case 3.—The patient was a girl, aged 13 years. In her case the eruption had been present for ten days. It began on the right thumb as a scratch, came up as a blister, and burst. Three days later the face became involved. I found a reddened circular area of the size of a sixpenny-piece over the metacarpo-phalangeal joint of the right thumb. On the face there were several slightly crusted, reddened areas of the size of hemp-seeds, some of which had coalesced, forming little patches each of the size of a finger-nail. These, the mother stated, had been originally vesicular. The child had been away in the country for a holiday with the patient in the following case.

Case 4.—The patient was a girl, aged 10 years. Her eruption had been present for ten days. It began as a white blister, containing "water," on the lobe of the right ear. There was a reddened, circular, dry area one inch in diameter on the dorsum of the left hand, a similar one about a quarter of an inch in diameter on the right wrist, and a few drying-up pustules about the occiput and the right ear. The pustular lesions of the occiput led me to examine for pediculi. I found numerous nits. This gave the key to the whole thing. There were no pediculi in the other children.

In connection with the above, I may mention that I have seen pemphigus neonatorum occur where the three other children of the family all had marked impetigo contagiosa of the ordinary type. I had no doubt in that case that the infant had contracted the bullous eruption from the other children.* I was asked to see the child because it was thought to be a case of congenital syphilis. I found that the family lived in one upstairs room, and the mother assured me that the house was quite healthy. I have also seen pemphigus neonatorum in sucklings whose mothers were suffering from abscess of the breast.

In the cases of bullous impetigo contagiosa which I have described above, the youngest child had best presented the bullous type, no doubt on account of the greater delicacy of the skin. In him the lesions looked like what is seen in subsiding pemphigus neonatorum, and the lesions had not the crusted appearances usually associated with ordinary impetigo contagiosa, whereas some of those in the older children had.

I am indebted to Dr. H. Radcliffe Crocker for kindly allowing me to make use of the cases.

(*Read November the* 15*th*, 1901.)

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* See also Matzenauer on "The Identity of Impetigo Contagiosa and Pemphigus Neonatorum," in the 'Wiener klinische Wochenschrift,' No. 47, 1900.

17. A CASE OF CONGENITAL UMBILICAL HERNIA.

By JOHN HOWSON RAY, Ch.M.

The following case is remarkable for the immense size of the hernia, and illustrates some of the difficulties in the treatment of this condition where the contained viscera are covered merely by peritoneum, some loose connective tissue, and the layer of amnion continuous with the cord. Strictly speaking, this is not a hernia, but a persistence and exaggeration of the condition which is normal about the end of the eighth week of fœtal life, where some intestine in relation to the yolk-sac still remains outside the abdomen, to be enclosed by the abdominal plates in the course of the third month.

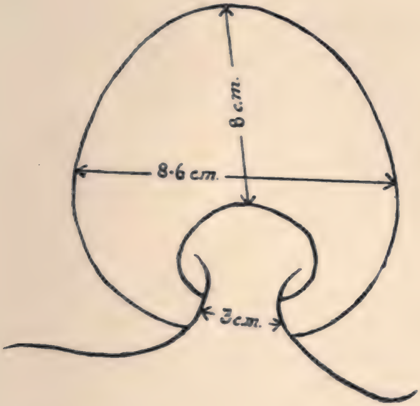
The infant, Ellen S—, aged 1 day, was admitted to the Manchester Children's Hospital by Dr. Ashby, on October the 11th, 1901, with an enormous umbilical hernia and evidence of bruising of the tumour during parturition.

The hernia was about the size of the infant's head, and was connected with the abdomen by a small pedicle, the hernia itself being globular and smooth, and having the cord attached a little below and to the left of its centre. The pedicle had a diameter of 3 cm., and was covered by healthy skin which terminated in a slightly raised flange-like margin, where it joined the amniotic structure covering the body of the hernia. The maximum horizontal diameter of the hernia measured 8·6 cm., the maximum vertical diameter was 8 cm., and the measurement from the edge of healthy skin to the pole of the swelling was also 8 cm. (see Fig. 3). Through the translucent amnion, in which several veins stood out distinctly, could be seen some effused blood, and this area showed a play of colours, including lymph-yellow, green, purple, blood-red, and patchy brown. The swelling was tense and tympanitic, and clearly contained some fluid and several coils of intestine.

The attached cord was flabby, wrinkled, and drying. The abdomen was moderately tense, the thorax well-developed, and the cardiac impulse normal. For the rest the infant was well-formed, well-nourished, and strong, and did not appear to be suffering severely.

Meconium passed on four occasions on the day of admission into hospital, and there was no vomiting. The temperature was 99° F., the pulse normal, and the respiration easy. Mr. Ray saw the case about twelve hours after its admission, and decided to wait before any operation was undertaken, if such a measure should be advisable. An antiseptic dressing was applied to the hernia. The following day the condition became worse, vomiting of green bile commenced, the temperature rose to 100·6° F., and obstruction became a marked feature. An operation was therefore undertaken about 4 p.m. on October the 12th, and on opening the sac and liberating some blood-clot and fluid blood it was found that there was recent peritonitis, with extensive adhesions between the coils of small intestine and the sac. The contents were found to be some omentum, about three feet of small intestine with a Meckel's diverticulum, the cæcum and appendix, and about six or eight inches of the first part of the colon.

FIG. 3.



The margins of the ring were sharply defined, especially above, and a finger could not be passed into the abdominal cavity whilst the gut was *in situ.*

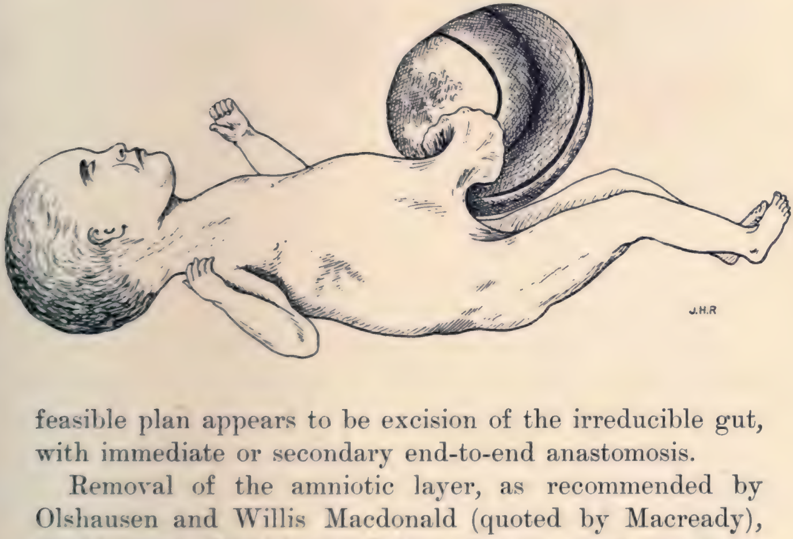
The adhesions were separated and two peritoneal bands within the sac were divided, and, after notching the ring above, a careful attempt was made to place the gut within the abdomen. A somewhat prolonged manipulation proving futile, the irreducible gut was clamped by means of rubber tubing, excised, and an end-to-end anastomosis carried out by circular suturing with mattress sutures passed on Lembert's plan outside. The anastomosis completed, a gauze drain was inserted just within the ring, and, the sac having been removed, the abdominal wall was closed by four sutures, including all the structures in the parietes. The condition of the infant gave evidence of much shock, but a hypodermic injection of strychnine and ether was given with good result, and recovery from the operation was marked in about an hour. The only food given consisted of whey and barley-water after the operation, but the vomiting persisted so that little was really retained until four hours later. On the day following the operation the bowels acted eight times, and vomiting again became a troublesome feature. The temperature varied from 100° F. to 101·8° F. The infant died of exhaustion about twenty-eight and a half hours after the operation. No post-mortem examination was permitted, but there was no distension of the abdomen at the time of death, and the child remained warm to the end.

The piece of intestine removed consisted of 72 cm. of small intestine and 6 cm. of cæcum and colon combined. The appendix measured 17 mm. in length, with an external diameter of 3½ mm. At a distance of 22 cm. from the ileo-colic junction a Meckel's diverticulum was found situated on the right or anterior face of the ileum. The diverticulum measured 22 mm. in length, and had an external diameter of 7 mm. at the attached base. The apex was continuous, with a solid cord—the obliterated vitelline duct—about 2 cm. long and 3 mm. broad, and attached to the sac about 1 cm. from the origin of the cord.

The sketch (Fig. 4) illustrating the case was made immediately before the operation.

As regards the treatment of such a severe form of so-called umbilical hernia, it is obvious that laparotomy will give the best chance, especially since this will include the removal of the infected outer covering of the sac; but the difficulty or impossibility of finding room within an abdomen not sufficiently developed to hold all the intestine makes it necessary to do something further, and the only feasible plan appears to be excision of the irreducible gut, with immediate or secondary end-to-end anastomosis.

Fig. 4.



Removal of the amniotic layer, as recommended by Olshausen and Willis Macdonald (quoted by Macready), would lessen the risk of infection, but the very thin layer of peritoneum remaining would seem to offer a very poor chance to the expectant line of treatment. In 'Diseases of Children,' by Ashby and Wright, two cases are mentioned where a portion of the liver protruded through a similar defect in the abdominal wall. In one case the sac and protruded liver sloughed and were dissected away, and the abdominal opening closed by sutures, but without success. In the other case the part was protected from irritation and left, but the child died. The authors mention a similar case by Underwood where recovery followed treatment by poulticing, and state that Tanner and others have had successful cases. No case closely resembling the subject of this communication appears to have been previously reported, and it would seem, therefore, that it is a case worthy of being placed on record.

(*Read November the* 15*th,* 1901.)

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18. CONGENITAL CYSTIC DEGENERATION OF THE KIDNEYS.

By W. GIFFORD NASH.

Specimen I was removed from a child, who died at the age of 6 months. The enlargement of the kidneys was noticed a few days after birth. The urine did not contain albumen, and the ureters and bladder were normal. There was also malformation of the large intestine.

The cæcum and ascending colon were small, the transverse colon dilated up to the splenic flexure, where there was a constriction, and the descending colon was also dilated.

Specimen II was removed from a child, who died at the age of 10 weeks. The enlargement of the kidneys was noticed a week after birth.

History.—The paternal grandmother of these children was pregnant thirteen times. In eleven instances a miscarriage or premature birth took place. One child died at the age of five months from bronchitis. The other, the father of these children, married an apparently healthy wife, and has had six children :

1. A male had a double inguinal hernia, and lived five weeks.
2. A male, living, with an inguinal hernia.

3. Born dead; a breech presentation with prolapse of the funis.
4. A male from whom *Specimen* I was removed.
5. Lived one hour.
6. Child from whom *Specimen* II was removed.

(*Exhibited November the* 15*th,* 1901.)

Discussion.

Dr. Theodore Fisher (Bristol) said there used to be a theory that the forms of cystic degeneration found in adults were congenital. He would like to hear from Mr. Nash whether it was frequent for children to live weeks or months with congenital cystic kidney.

Mr. W. G. Nash (Bedford), in reply, said he was under the impression that some of the cases lived to adult age, and believed there were instances on record of patients arriving at twenty or thirty years of age. When looking up the history of such cases he saw reference to three consecutive children in one family suffering from the condition.

19. A CASE OF PULMONARY REGURGITATION.

BY EDMUND CAUTLEY, M.D.

INCOMPETENCE of the pulmonary valves, leading to regurgitation, is rightly regarded as one of the rarest forms of valvular lesions. It is usually associated with endocarditis of the infective variety, or with pulmonay stenosis. In an article on the subject by Newton Pitt in Allbutt's 'System of Medicine,' the writer refers to ninety-nine cases, verified post mortem. More than half of these were due to infective endocarditis. The chief other causes enumerated are pulmonary stenosis, abnormalities of the pulmonary valves, dilatation of the pulmonary artery, and pressure of an aortic aneurysm on the pulmonary valves. Of the fifty-seven cases associated with infective endocarditis no obvious cause was found in nineteen, but in eight instances there was congenital pulmonary stenosis. As regards age, only two of these fifty-seven cases were in children under ten years of age, and twelve in patients between ten and twenty years of age. The present case is an illustration of pulmonary regurgitation in a girl aged fifteen years, secondary to pulmonary stenosis, and associated with acute endocarditis of the valves.

A girl, aged 15 years, was admitted into the Metropolitan Hospital under my colleague, Dr. Oswald Browne, on May the 7th, 1901, and was discharged on May the 23rd. She subsequently attended my out-patient clinic, and was readmitted to the hospital under my care on August the 13th, and died on October the 10th, 1901. As regards the family history, it was stated that her parents and two brothers were alive and healthy; the mother had had rheumatic fever three times; she had lost several brothers and sisters, but none had died from rheumatic fever. She was said to have been very small as a baby, blue and cold. Morbus cordis was diagnosed at the age of one and a half years. At various periods since April, 1898, she came under my care, and on examination was found to have a marked systolic thrill and murmur in the pulmonary area. Two years before admission she was in the London Hospital for rheumatic fever. After this she had diphtheria, and was in the Fever Hospital at Homerton. She had been subject to joint pains, and she had worked as a feather curler. On May the 7th she was admitted for headache, vomiting, and general debility, with a temperature of 103·4° F. The patient had a high colour and was well-nourished; there was no clubbing of the extremities or œdema. The apex of the heart could not be localised, and there was no thrill. Dulness extended to the middle of the sternum, to the third left intercostal space, to the left anterior axillary line, and down to the seventh rib. The sounds at the aortic base were clear and ringing. At the pulmonary base the first sound was clear, while following the second was a diastolic murmur conducted downwards, and heard in the third and fourth left intercostal spaces close to the sternum, being loudest lower down. A systolic murmur was also heard at the apex, in the axilla, and faintly behind. The pulse was 96 beats to the minute, regular, not sudden, of fair volume and good tension. A few moist sounds were heard at the base of the left lung. No enlargement of the liver was detected. The urine was normal. The temperature fell steadily to normal in two days. In a week she was allowed up, and on May the 23rd she was sent to a convalescent home.

The patient remained in her usual health until August the 13th, when she complained of pain in the right knee, and minute bright red spots were seen on the legs. On coming to the hospital in the afternoon she seemed to be very ill, and was admitted. Her colour was high with a slight tendency to duskiness of the cheeks. The temperature was 101·6° F., and the pulse was 108 beats to the minute, regular, and of good volume. The lungs were normal, except for a few crepitations at the bases. The apex of the heart was in the sixth space in the nipple line ; the impulse was not heaving. Diffuse pulsation extended for from two to three inches to the right of the sternum. There was marked epigastric pulsation. There was no thrill anywhere. The first sound at the apex was rather rumbling in character. Both sounds at the base in the second right and left intercostal spaces were clear and ringing. In the third and fourth left intercostal spaces near the sternum was a diastolic murmur, which increased in intensity on expiration and in the upright posture. After being a few hours in bed the pulse-rate fell to 84 to the minute ; in character the pulse was small, soft, and not sudden. There was no evidence of enlargement of the liver and spleen. No clubbing of the extremities was present. On a light diet and small doses of salicylate of soda the pain in the knee disappeared in two days, and the pyrexia subsided in four days. On August the 16th moist sounds were abundant at both bases of the lungs behind. There was no cough. On the 20th the diastolic murmur was louder, and was heard at the base and down both sides of the sternum. The pulse was small and rather sudden. On the 24th there was pain in the right side on breathing, which was probably pleuritic, though no definite friction was heard. For the next week she had a moderate amount of fever in the evenings. On the 26th the temperature rose to 103·4° F. Nothing was found to account for the fever, except the crepitations at the bases. In other respects she felt well. Up to September the 5th for a week she had been practically free from fever. That morning she brought up three or four pints of blood, becoming collapsed, restless, and covered with sweat; the pulse was almost imperceptible. On the 6th she vomited some altered blood during the night. The pulse-rate was 96 to the minute. On the 21st the temperature had remained down, and there had been no more hæmorrhage. She had been on full diet for two days and allowed to sit up in bed. On the 24th she was up in a chair. On the 26th more hæmorrhage occurred. Although she had been on liquid or semi-solid food, and seemed to progress favourably, being bright and cheerful and free from fever, she had another attack of hæmorrhage on the night of October the 5th, and on the night of the 6th she brought up about three ounces of blood. On the 7th she brought up a few clots on vomiting in the morning. The heart was more dilated, the apex being felt an inch outside the nipple line in the sixth space. At the apex there were systolic and diastolic murmurs, the former not being conducted into the axilla. A marked diastolic thrill was now felt over the left border of the sternum, and the diastolic murmur was very much louder. On the 10th she brought up over a pint of blood with vomiting, became very pale and collapsed, and died quietly in a few minutes. The temperature was normal or subnormal during the last two weeks of her life.

Necropsy.—At the post-mortem examination rigor mortis was present. A little blood was exuding from the nose. The trachea contained frothy blood. A little blood-stained fluid was present in the left pleural cavity. The left lung weighed sixteen and a half ounces; a large infarct filled up about one eighth of the lower lobe, and the blood-vessel immediately above this contained an ante-mortem clot. Many smaller infarcts were also present in this lobe. The right lung weighed nine ounces; there were many pleuritic adhesions, and many small infarcts were present in the lower lobe. The pericardial cavity contained about an ounce of clear serum. The heart weighed eleven ounces, and was very firm and globular; the right side was much hypertrophied. The pulmonary valves were much thickened and puckered. On two of the valves were large warty vegetations with apparent loss of substance. The right ventricle was dilated, its walls being much hypertrophied, and the musculi papillares were much enlarged. All the other valves were normal. The liver weighed thirty-one ounces, and showed commencing "nutmeg" change; the spleen weighed six and a half ounces, and the kidney six ounces. No infarcts were found in any of these organs. The stomach contained nearly two pints of frothy blood. Blood was present in the duodenum. No ulceration of the mucous membrane was found.

Remarks.—The case presented all the classical signs of the lesion. The cardiac apex was displaced downwards and outwards. There were marked dilatation and hypertrophy of the right ventricle and epigastric pulsation. In addition there was a diastolic bruit heard in the third and fourth left intercostal spaces, close to the sternum, a bruit which might easily have been ascribed to aortic regurgitation, but the typical water-hammer pulse of the latter lesion was absent. Further, the murmur was intensified on expiration and in the upright posture. Emboli were carried to the lungs and produced frequent attacks of hæmoptysis. Such hæmoptysis is said to be present in one third of the cases, chiefly in those due to infective endocarditis. A capillary pulse has been described as occurring in the pulmonary circulation—*i. e.* a jerky vesicular murmur, waxing and waning with the cardiac contractions. It was not detected in this case.

It is a curious fact that although pulmonary stenosis is a common lesion it is rarely associated with incompetence, whereas the reverse holds good in aortic lesions. This can be accounted for by the low blood-pressure in the pulmonary circulation not creating a regurgitant stream sufficiently powerful to produce a murmur, even if the valves are incompetent.

The case presented many difficulties from a clinical point of view. The physical signs were those of pulmonary regurgitation, associated with a high complexion suggestive of some pulmonary stenosis, and a variable pyrexia which might be due to rheumatism, infective endocarditis, or pulmonary complications. On the other hand, there was no evidence of pulmonary stenosis, such as clubbing of the extremities and a systolic murmur or thrill in the pulmonary area. The rarity of pulmonary regurgitation made one think that a more probable diagnosis was that of aortic regurgitation, but the absence of a characteristic pulse, the absence of signs of hypertrophy and dilatation of the left ventricle, and the presence of signs of dilatation and hypertrophy of the right ventricle were opposed to such a view. In addition, it would have been necessary to assume the presence of some congenital lesion to account for the high colour. Again, the history of past rheumatic fever and the presence of pain in the knee suggested that the aortic lesion was the more probable, for rheumatic endocarditis rarely affects the pulmonary valves. In fact, in cases of pulmonary regurgitation a past history of rheumatism has only been recorded in about 10 per cent.

Another possible diagnosis was that of mitral stenosis associated with adherent pericardium. In favour of this was a note when the child was first admitted in May that the sounds "seemed a little sticky." The murmur was, however, too characteristic of regurgitation through the aortic or pulmonary valves, and the characters of the pulse were opposed to such a diagnosis.

Infective endocarditis was suggested as a cause on account of the pyrexia and the unusual physical signs.

Moreover, it is stated in text-books that a diastolic pulmonary murmur is not heard except in the presence of infective endocarditis. On the other hand, it must be pointed out that the murmur was present for many months and while the patient was apparently in good health. The pyrexia was not serious or constant, and there was no fever at all during the last fortnight. The purpuric spots were a little in favour of infective endocarditis, but they were few and soon disappeared. There were no albuminuria, no diarrhœa, and no splenic enlargement. The hæmorrhage presented difficulties; it was, no doubt, from the lungs, but it was always accompanied by vomiting, and ascribed by the house physician who saw it to hæmatemesis. If it had come from the stomach it could be explained as the result of cardiac failure and secondary passive congestion of the stomach.

Was the case one of infective endocarditis? On the one hand, this is the usual cause of the condition. On the other hand, the diastolic bruit was present for at least five months before the fatal issue, and the infarcts in the lungs showed no signs of breaking down. Possibly the endocarditis was rheumatic in origin.

(*Read November the* 15*th*, 1901.)

Discussion.

Dr. Theodore Fisher (Bristol) thought the case was one of chronic infective endocarditis. The variety of lesions presented by that condition was known to be great. In some cases one might see there had been extensive ulceration which had healed, only one or two vegetations remaining to betray the acute nature of the disease. With regard to the infarcts, it did not necessarily follow that those in ulcerative endocarditis would suppurate. In some cases of death from infective endocarditis one could see, side by side, simple infarcts and suppurative infarcts, as well as the scars of former infarcts. Although well-marked right-sided endocarditis is generally infective in nature, one not uncommonly may see, in death from rheumatic pericarditis and fatal cases of chorea, small characteristic rheumatic vegetations on both the pulmonary and tricuspid valves. Those valves undoubtedly, like the valves on the left side of the heart, may be affected by rheumatism, but the lesions rarely become chronic.

Dr. Sansom expressed his gratitude to Dr. Cautley, and, on the whole, endorsed the view expressed by Dr. Theodore Fisher. He had never seen a case exactly like it, and he believed it would have been practically impossible to make a diagnosis during life. But taking the physical with the rational diagnosis there would be a little more hope of getting at the truth. No doubt the hæmoptysis was due to infarction of the lung, and that was the salient feature of the case, though it occurred comparatively rarely in aortic cases, being more common in disease of the right heart. He agreed with Dr. Fisher that it would be a mistake to say the thrombi would necessarily be infective, and they often left little or no trace behind. It was very common in really infective endocarditis to see, in the same lung, recent and apparently uninfective emboli, some old ones which certainly were not, and some which possibly might be infective. It was not common to be able to demonstrate an infective embolus. It was known that a systolic murmur indicative of mitral regurgitation would occur in the presence of aortic disease, and therefore the whole case might have been explained before the autopsy on that ground. He had recorded several cases in which the temperature chart afforded no guidance necessarily, the temperature having been normal or subnormal. But, looking at the matter all round, he endorsed the opinion expressed by Dr. Theodore Fisher. The specimen seemed to show loss of tissue besides the large vegetations, which were more common in infective endocarditis. He believed it to be a case of slow-growing infective endocarditis, and therefore differing from the true rheumatic form, and that it vented its chief action on the pulmonary valves. The case was most instructive.

Dr. Cautley, in reply, said the hæmoptysis was not so definite as Dr. Sansom gathered; it was difficult to say whether it was hæmoptysis or hæmatemesis. It had been explained on the assumption that there was cardiac failure. It was suggested also that there might be a secondary gastric ulcer. He agreed as to the endocarditis. He thought the case started as a congenital pulmonary stenosis, and that later, as a result of rheumatism or some infective process, the valves gradually disintegrated, and there had been some form of endocarditis which was limited to the pulmonary valves.

20. OBSERVATIONS ON THE ÆTIOLOGY AND MORBID ANATOMY OF TUBERCULOUS MENINGITIS.

By EDMUND CAUTLEY, M.D.

For the data of this paper I have taken the records of the last twenty-seven cases of tuberculous meningitis which have been under my care during life, and have been subsequently examined in the deadhouse. Though the number is too small to justify dogmatic statements on any one point, the results obtained from the inquiry are sufficiently definite to enable me to bring before you certain deductions as to the ætiology of the disease.

1. *The Age Distribution.*

Age in years.	Males.	Females.	Total.
0— 1	0	3	3
1— 2	5	1	6
2— 3	2	3	5
3— 4	1	3	4
4— 5	2	2	4
5—10	4	1	5
	—	—	—
	14	13	27

No less than twenty-two out of the twenty-seven cases occurred during the first five years of life, and only five during the second five years. This result confirms those of other observers that the disease is most common under five years of age. Some writers state that it is rare during the first year, and give statistics in support of their statements. Others find it frequent during the first year. It is not always clear, from the figures quoted, whether the statements are based on cases diagnosed during life, or only on those which have been verified after death. If the figures are based on clinical records only, it is probable that the frequency of the disease during the first year would be over-estimated on account of the difficulty in invariably making an accurate diagnosis between the simple posterior basal meningitis, which is unduly prevalent during the first year of life, and true tuberculous meningitis. My own figures show that the distribution of the cases during these first five years is fairly equal for the different years. There is no preponderance of either sex; fourteen males to thirteen females.

2. *The Influence of Heredity.*

In recent years it has become customary to ascribe very little importance to heredity in the production of tuberculous affections, except in so far as the sufferers are exposed to direct infection from association with a tuberculous relative, and may inherit a peculiarity of constitution which renders them susceptible to such infection. It is well known how difficult it is in hospital practice to obtain accurate family histories, and that all statements as to the occurrence of "consumption" in the parents or other relatives must be viewed with suspicion. The results obtained on this point are opposed to the theory of heredity, but must not be too strongly insisted upon. In twenty-two of the cases it was definitely stated in the notes that there was no history of phthisis in the parents or other children. Of the remaining five cases a few facts may be given.

In only one case was the mother stated to be affected.

Case 14.—A female, aged 6 months. The mother was said to be very ill with "consumption and bringing up blood." The child was only nursed for one week, and was then brought up on condensed milk. She had been taken out of doors very little. She died with symptoms of tuberculous meningitis, and there were found, in addition, tuberculous consolidation of the upper lobe of the left lung, a cavity in the upper lobe of the right lung, general dissemination throughout the lungs, and a few tubercles in the spleen. No note was made as to the condition of the mediastinal or mesenteric glands.

No better instance could be given of probably direct infection from the mother through the respiratory tract.

The next four cases also illustrate the possibility of direct infection from a parent.

CASE 2.—A female, aged 3 years 9 months, died after an illness of nineteen days' duration. Tubercles were found in the meninges, liver, and spleen. No caseous glands were found. The cribriform plate of the ethmoid was carious.

While the child was ill the father was an in-patient at the Brompton Hospital for Consumption. The case suggests strongly that infection took place through the nose. To quote Osler, "in those instances in which no primary focus has been discovered it has been suggested that the bacilli reach the meninges through the cribriform plate of the ethmoid from the upper part of the nostrils, but this is not probable." It is certainly not probable that the meninges can be infected by this route unless disease of the bone is first set up.

The possibility of direct infection is present in the next case, which also presents another point of interest, to be subsequently discussed, namely, the influence of injury in the production of the attack.

CASE 1.—A female, aged 2 years 6 months, died after an illness of seventeen days' duration. After death there was found basal meningitis, with only a few tubercles in the meninges, and no evidence of a tuberculous focus. A few pleural adhesions were present at the apex of the right lung. The child had a fall three weeks before the onset of the symptoms. The father was said to be "consumptive," and a sister was at the time in the Brompton Hospital for the same disease. It is a question whether the tubercles in the meninges were the primary cause, or secondary to a basal meningitis set up by the fall.

The other two cases presented interesting features. In both the father was said to be "consumptive."

Case 19.—A male, aged 2 years, died after an illness of eighteen days' duration. A large tuberculous cavity was found in the lower lobe of the left lung, together with general dissemination of tubercles throughout the lungs, and caseous mediastinal glands.

Case 24.—A male, aged 3 years 7 months, developed left hemiplegia during the course of the illness. An old tuberculous mass was found in the right external capsule, and there were tuberculous nodules in the cerebellum. The lungs were stuffed with tubercles, and the mediastinal glands were caseous. Only one tubercle was present in the spleen.

These five cases were the only ones in which a family history of tuberculous disease was obtained, and they all illustrate the probability of direct infection.

3. *Injury as an Ætiological Factor.*

In only one instance (Case 1), that already referred to, could injury be invoked as the exciting cause of the disease. Even in this case it is possible that the meninges had become infected before the injury, for the presence of pleuritic adhesions at the right apex suggests former tuberculous mischief. All text-books recognise the importance of this point. It is more specially applicable in the case of children who go to school. For some reason or other the child receives a box on the ear, or a rap on the head by the knuckles or ruler, or other punitive instrument in the hands of the teacher. Subsequently the child dies from tuberculous meningitis, which is ascribed by the parents to the injury. Possibly the unfortunate teacher is hauled before a coroner's court and severely censured by a typical British jury. The common and usually accepted explanation is that the child was punished for stupidity or inattention, the state of mind so likely to be present during the early stages of the disease. It is, therefore, imperative in such cases that a careful *post-mortem* examination should be made in order to ascertain whether there is a tuberculous focus present which might have given rise to the meningeal infection. On the other hand, a case like the one quoted might be brought forward as an argument in favour of injury being the exciting cause. Injury to the brain or meninges might cause local congestion or damage of such a nature as to render the affected part a suitable soil for the growth of the tubercle bacillus, which without such advantageous conditions might have perished. This is the only instance out of the twenty-seven cases which is favourable to this view.

4. *The Channel of Entrance of the Bacillus into the System.*

In view of the recent discussions and observations on the relationship of the bacillus of bovine tuberculosis to that of the human disease, and on the infectivity of the milk of tuberculous cows, this point is one of extreme importance. In the last edition of Fagge's 'Text-book of Medicine' (vol. i, 1901), edited by Pye-Smith, it is stated in the chapter on tuberculous meningitis that "the exciting cause is the entrance of Koch's bacillus into the circulation, and in children this is probably by way of the intestinal tract from the milk of tuberculous cows." Kanthack, Delépine, and others have proved that the bovine tubercle bacillus is frequently present in cow's milk. It has been found by Bang, Bollinger, and others in the milk of cows affected with general tuberculosis or tuberculous disease of the udder. It has been experimentally determined by Hirschberger that the milk of tuberculous cows may be infective, even though the udder is unaffected. Kempner and Rabinowitsch in 1899 found the bacillus in the milk of cows which reacted to tuberculin, but gave no other evidence of tuberculosis; and their results have been confirmed by Ostertag, Adami, and Martin. There is, in fact, no shadow of doubt that the bovine tubercle bacillus is often present in cow's milk. One warning must be given on this point. Acid-resisting bacilli, similar in nature and appearance to the tubercle bacillus, have been also found in milk (Petri, Beck, Santoni, Rabinowitsch, etc.), in the udder (Ludwig Neufeld), in the intestines of healthy and tuberculous cows (Olt), in the Timothy-grass sometimes used for fodder (Moeller), and in the dung (Severin). These bacilli cannot be distinguished microscopically, but can be readily distinguished by intra-peritoneal injection into guinea-pigs. Tuberculosis can be produced in calves and pigs by feeding them on tuberculous milk; and, by analogy, it has been assumed that a similar result will arise in children. In support of this the reports of the Registrar-General are often quoted. In these reports it appears that the disease called tabes mesenterica, usually regarded as a tuberculous affection, is increasingly prevalent among infants. With regard to this latter statement I wish to enter a very strong protest. Tabes mesenterica is very rare among infants, and there is little doubt that almost all the cases so entered in mortality statistics, as occurring in infants under one year of age, are neither more nor less than the interesting disease known to the public by that blessed name "consumptive bowels," that is diarrhœa and wasting due to improper feeding and ending in death. I have never yet seen a true case of tabes mesenterica in an infant examined *post mortem*, although I have been constantly on the watch for one, and I am strongly convinced that it would conduce to scientific accuracy if the name were eliminated from our nosology.

To return to the point under consideration. The main channels of infection are the respiratory and the alimentary tracts. Of quite secondary importance are the ear and skin, both of which occasionally prove channels of infection. Let us consider, then, what evidence these *post-mortem* records yield in favour of one or other of these modes of entry of the bacillus.

Using the term mediastinal glands to include all the glands in the anterior and posterior mediastinum, at the bifurcation of the trachea, and at the roots of the lungs, a most striking fact is at once revealed. In only two instances were these glands normal, and in these the tuberculous process was limited to the base of the brain, being in one of them associated with caries of the cribriform plate of the ethmoid, and in the other possibly secondary to a localised pleurisy at the apex. In two other instances (Cases 14 and 16) no note was recorded as to the condition of these glands, but I have little doubt that, considering the advanced tuberculous disease in the lungs, the glands must almost certainly have been affected. In the remaining twenty-three cases one or more of the mediastinal glands was caseous. The one most often affected, frequently the only one, was the gland situated at the bifurcation of the trachea, commonly called the pre-tracheal gland. In four of these twenty-three cases the mesenteric glands were also caseous. In one of these four (Case 4) the peritoneal surface of the diaphragm was covered with miliary tubercles, and there were tubercles in the spleen, while the lungs only showed evidence of bronchitis. The other three cases, in which both sets of glands were caseous, are of much interest.

Case 25.—A male, aged 13 months, was under my care for two months for subcutaneous tuberculous nodules. There were four such nodules, varying in size from a split pea to a horse-bean, one of which was said to have been in existence since the age of four or five months. He developed meningitis, from which he died. In addition to tuberculons basal meningitis, the brain was found to contain caseating nodules, the lungs showed general dissemination, especially at the right apex, and there was extensive caseation of the mediastinal and mesenteric glands, with a few tubercles in the spleen.

In the other two (Cases 26 and 27) there was evidence to warrant the assumption that the abdominal tuberculosis was secondary to the swallowing of tuberculous material coughed up from the lungs. Thus, in a boy aged 17 months, tuberculous vomicæ were found in both lungs and two small tuberculous ulcers in the small intestine. In a boy aged two years, an old caseating and calcifying nodule was found in the lung, but, unfortunately, the intestines were not examined.

In nine instances there was more or less dissemination in the lungs, or in the lungs and pleura, but no evidence of old standing disease. In only one of the nine, the child with subcutaneous nodules, were the mesenteric glands caseous.

In twelve instances the lungs showed extensive tuberculous disease of old standing, namely, caseation, and in some cases excavation.

Looking at the facts from another point of view, it seems somewhat remarkable that, if the alimentary canal is so liable to tuberculous infection as the upholders of the theory of infection by tuberculous milk would have us believe, in only two out of these twelve cases in which the disease was advanced, and of old standing in the lungs, was there evidence of infection of the mesenteric glands. In two the state of these glands was not noted. Of course it is clear that such infection may have been present, and that microscopical or experimental evidence might have proved some of these glands infected; but even if this were so, the facts strongly confirm the view that the respiratory tract was the primary source of infection. It is also clear that the alimentary tract is not markedly susceptible to infection by the human tubercle bacillus.

It has been stated that the tubercle bacillus of cattle is not dangerous to human beings. Even if we only accept the statement in a modified form, and say that the bovine bacillus is not nearly as infective as the human bacillus, our *post-mortem* facts afford a still stronger proof of the insusceptibility of the alimentary tract to infection by the bovine bacillus, seeing that it so often escapes, although exposed to the risk of infection by the human bacillus in chronic tuberculous disease of the lungs. In young children, as is well known, sputum is swallowed and not expectorated. Consequently the liability to secondary infection of the alimentary canal is enormous, whereas the *post-mortem* evidence proves it to be infrequent.

In six instances was there no evidence of tuberculous disease of the lungs, and yet in four of these the mediastinal glands were caseous. In one of the four the mesenteric glands were also caseous. It is possible that in these cases a small tuberculous lesion may have healed and left no trace visible to the naked eye, or that the bacillus reached the glands through some other channel.

In some instances in which the lung was affected the question arose whether the lung mischief was primary or secondary to an extension from the glands. In one case it was clear that the main part of the lung disease was due to the actual rupture of a breaking-down gland into its substance (Case 23). In some others the tuberculous process could be seen extending backwards from the glands along the lymph channels in a radiating fashion into the lungs.

The probable sequence of events in most cases is that the bacillus is carried by the blood or lymph stream to a congested gland, and there develops, finally causing caseation. Such a caseous gland may remain quiescent, may become calcified, may break down, may ulcerate through into the trachea or œsophagus, or may rupture into the lung or pleural cavity. In any case, if it breaks down the infective material may become widely disseminated and cause a general infection.

The *post-mortem* evidence may be summed up shortly as proving that the respiratory tract is the great channel of infection. It therefore follows that the danger from swallowing tuberculous cow's milk has been much exaggerated.

It is sometimes asserted that the bacillus can get into the system through the alimentary canal without producing a local lesion. Caseous mesenteric glands are found, although the mucous membrane of the intestines appears normal. If the bacillus does not pass out of the intestines through the lymph channels to the glands, it might possibly pass into the blood-stream. Practically all the blood from the intestines goes through the liver. We should expect, then, that if the bacillus passed into the blood-stream it would produce tuberculous disease of the liver. Miliary tubercles are indeed found in the liver, more often in the spleen, and less often in the kidneys, but only when there is considerable general dissemination throughout the body. It is rare to find old tuberculous foci in the liver, and in none of these cases was such a focus found. There is no evidence at all strongly in favour of infection of the blood-stream through the intestinal tract.

5. *Evidence derived from the Diet of the Patients.*

In eight out of thirteen infants under three years of age the child was brought up on the breast for the greater part or all of the first year of life. In four the mode of feeding was not stated. In one the child was brought up on condensed milk, for the mother was too ill from tuberculous disease of the lungs to nurse it. These facts, as far as they go, are suggestive that cow's milk is not a very virulent source of infection, for a very large number of infants are brought up on cow's milk. During the last ten years I have brought up a large number of children on cow's milk, and as far as I know not one of them has subsequently come under my care for tuberculous meningitis. The milk has been boiled, if the mothers have carried out the directions given.

6. *The State of the Brain as indicative of the Prospect of Operative Treatment proving Beneficial.*

In about one third of the cases there was no excess of fluid in the ventricles. In the remainder the fluid varied from a slight excess to a large amount. In many instances the cerebral tissues were very soft and occasionally almost diffluent. Operative treatment can only be indicated as a means of relieving pressure, and under the assumption that the child is dying from the coma induced by pressure. Such is not always the case, for many children died comatose in whom there was no evidence of increased intracranial pressure after death. The coma is probably due to the softening of the brain, which may be associated with excess of fluid in the ventricles or may occur quite independently. Hence it is clear on anatomical grounds that operative measures would only relieve pressure in about one third of the cases, and that even in these it would do little or no good, on account of the softening so often present and dependent upon insufficient blood-supply rather than on excess of fluid. Moreover these records indicate how very rarely the disease is limited to the brain, and that the operation would only afford temporary alleviation of symptoms.

7. *Classification.*

It is possible to divide the cases on anatomical grounds into three groups :

1. Simple tuberculous meningitis, in which the disease is limited to the brain ; very rare.
2. Tuberculous meningitis, secondary to a localised tuberculosis with little dissemination. The child dies from the meningitis.
3. General miliary tuberculosis : cases in which the meningeal symptoms bear a comparatively unimportant relationship to the disease.

To conclude, I am strongly convinced that inheritance means exposure to infection of a weakly or predisposed child ; that injury is very rarely an exciting or predisposing cause ; that the respiratory tract is the great channel of infection ; that the alimentary tract is rarely primarily infected ; that cow's milk is very rarely, if ever, the source of infection ; that limitation of the tuberculous process to the meninges is very rare ; that the prognosis is very hopeless on account of the extent of the tuberculous disease elsewhere ; and that operative treatment may be discarded as experimental rather than useful.

(*Read November the* 15*th,* 1901.)

No.	Sex.	Age.	Pleura.	Lungs.	Glands.		Other lesions.
					Mediastinal.	Mesenteric.	
1	F.	2.6	A few adhesions at right apex	No tubercle	Normal	Normal	A few tubercles at the base of the brain. No other evidence of tuberculosis.
2	F.	3.9	Normal	„	„	„	Caries of the cribriform plate of the ethmoid.
3	M.	1.1	„	„	Caseous	„	
4	F.	3.6	„	„	„	Caseous	Peritoneal surface of the diaphragm covered with miliary tubercles.
5	M.	4.6	„	„	„	Normal	
6	M.	9	Old adhesions of left lower lobe	„	„	„	
7	F.	2	Recent adhesions on right side	Scattered miliary tubercles in right upper and middle lobes	„	„	Broncho-pneumonia of left lower lobe.
8	F.	4.6	A few tubercles on right pleura	A few tubercles in right lower lobe	„	„	
9	F.	1.6	A few tubercles on both parietal pleuræ	General dissemination	„	Large	A few tubercles on parietal layer of the pericardium.
10	F.	4.6	Tubercles scattered over both pleuræ	General dissemination	„	Normal	
11	M.	4.9	Old adhesions at right apex	Stuffed with rather advanced miliary tubercles	„	„	Liver and spleen large; both contain tubercles. Perisplenitis.

No.	Sex.	Age.	Pleura.	Lungs.	Glands.		Other lesions.
					Mediastinal.	Mesenteric.	
12	M.	5	Normal	General dissemination	Caseous	Normal	
13	F.	8	”	A few tubercles scattered throughout both lungs	Very extensively caseous	”	Acute endocarditis.
14	F.	0.6	”	Tuberculous consolidation of left lower lobe. Cavity in right upper lobe. General dissemination	Not noted	Not noted	
15	F.	0.6	”	Caseous patches in right middle and lower lobes. General dissemination	Caseous	Normal	
16	F.	0.11	”	Caseous mass and cavity formation in right lower lobe	”	”	
17	M.	1.4	Right lower lobe universally adherent	Extensive caseation of right lower lobe	”	”	Large liver.
18	M.	1.6	Pleuritic adhesions at right base	Caseous mass at base of right lower lobe	Not noted	Not noted	
19	M.	2	Pleuritic adhesions	Cavity in left lower lobe and tubercles in the rest of the lung; a few in the right lung	Extensively caseous	Normal	
20	F.	2.10	Recent adhesions of right upper lobe	Caseous patch in right upper lobe, beginning to break down	Caseous	Large	

No.	Sex.	Age.	Pleura.	Lungs.	Glands.		Other lesions.
					Mediastinal.	Mesenteric.	
21	F.	3	Patch of tubercles on right parietal pleura and adhesion to diaphragm	Extensive tuberculous broncho-pneumonia	Caseous	Large	Tuberculous cholangitis. Perihepatitis. Perisplenitis.
22	M.	6	Normal	Old fibroid change; recent tubercles and caseous masses in right upper lobe. Caseation of both lower lobes, spreading from glands	Very extensively caseous	Normal	
23	M.	7	,,	Caseous right upper lobe due to rupture of a caseous gland into it	Caseous	,,	
24	M.	3.7	,,	General dissemination	,,	,,	Old tuberculous mass in right ext. capsule. Tuberculous nodules in cerebellum.
25	M.	1.1	,,	General dissemination, especially at right apex	,,	Caseous	Tuberculous subcutaneous nodules. Four caseating nodules in the brain.
26	M.	1,5	,,	Small vomicæ in right upper and left lower lobes. General dissemination	,,	,,	Two small tuberculous ulcers in the small intestine. Perisplenitis.
27	M.	2	,,	Caseating and calcifying nodule in right middle lobe	,,	Very extensively caseous	Intestines not examined.

Discussion.

Dr. Sutherland agreed with the views expressed by Dr. Cautley, and particularly as to the statements in the current text-books about the prevalence of tabes mesenterica in early life, for the deaths were mostly registered under that title without any examination having been made. With regard to the alimentary canal, if the child had been properly fed he thought the risk of tuberculous infection in that way was not great.

Dr. Theodore Fisher (Bristol) said that at a dairy near New York forty cows were found to be tuberculous, and they were all killed; but of the hundreds of children who had consumed that milk, only one was tuberculous, and it was not certain that it came from that source. His own experience fully bore out the remarks of Dr. Cautley.

Mr. Willmer Phillips (Southsea) asked whether Dr. Cautley had had any experience of tubercle having been disseminated through carious teeth. He had suspected such a source, quite apart from alveolar abscess. He thought one case of tuberculous meningitis which came under his notice might have occurred in that way.

Dr. Cautley, in reply, said he had no evidence of tuberculous trouble starting from carious teeth. In every case he found definite sources elsewhere. In one instance where the disease was limited to the meninges there was caries of the cribriform plate of the ethmoid; and in another similar case it probably followed injury or infection from an old pleurisy at the right apex. There might be tuberculous lesions in the mouth, but he included them among skin lesions.

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21. A CASE OF PAROXYSMAL HÆMOGLOBINURIA IN A CHILD OF THREE YEARS.

By GEORGE CARPENTER, M.D.

Elsie W—, aged 3 years, was brought to the Evelina Hospital on January the 16th, 1902. She is a pale child, with a full face on which are seated two lesions of molluscum contagiosum. She has a muco-purulent discharge from the nostrils, probably due to post-nasal adenoids. With her was brought a bottle of port-wine-coloured urine which is here for inspection; it gives the hæmoglobin reactions, and there are no red blood-corpuscles in it.

She attended the out-patient department in May, 1901, for abdominal pains of sudden onset, of about three minutes' duration, during which she screamed; the pains were said to have been induced particularly by cold water though not necessarily. The urine was at one time "thick," at another "clear;" there was no blood, but a sediment of "little red lumps" had been noticed. Her temperature then was 103° F., and her spleen a trifle enlarged. At this period an infant sister was being treated at the hospital for congenital syphilis.

There is a history of tubercle on the father's side for two generations back, and also one of rheumatism in the family.

On this occasion, the second visit, she is stated to have been much better until two nights previously, the night of the 14th inst. She then passed port-wine-like urine. On the 15th she passed similar coloured urine two or three times, a sample of which is now shown. By the night of the 15th the urine was much clearer, and on the morning of the 16th it was quite clear, as is now the case. She was feverish, restless, and fretful during the attack. I cannot find any evidence of congenital syphilis in her case. Her spleen is now natural. The extremities are warm, and there is no suspicion of Raynaud's disease.

(*Exhibited January the* 17*th*, 1902.)

Discussion.

The Chairman (**Mr. A. H. Tubby**) asked whether a spectroscopic examination of the urine had been made.

Dr. Sutherland asked whether there were any oxalates in the urine. He had usually found such present.

Mr. Pernet asked whether the patient had ever been out of this country.

Dr. Macalister (Liverpool) said that he was struck by the circumstance that the child had a chronic nasal discharge, and the mother gave a history of frequent feverish attacks. It is known that a considerable number of absorption fevers are related to chronic affections of the nasal passages, and he could well imagine that disease of the nose such as was present in this child might induce a perverted blood condition resulting in hæmoglobinuria.

Dr. Leonard Guthrie said that although there seemed to be no evidence of specific disease in the case, the history of such a taint in the other child was presumptive evidence of syphilis operating in the present instance. He remembered having two sisters in the same family who undoubtedly were syphilitic, and both of them had paroxysmal hæmoglobinuria.

Dr. Nabarro said he had examined the urine for Dr. Carpenter with the spectroscope, and it showed two strongly marked absorption bands between the Fraunhofer lines D and E in the yellow and green, a band in the red, and partial absorption at the red and violet ends of the spectrum.

Dr. George Carpenter, in reply, said the chronic nasal discharge had been noticed, and was due to post-nasal growths. He had not examined the urine for oxalates in excessive quantities, and he did not see any crystals when making a microscopical examination. The patient had not lived abroad. She was anæmic. He said he remembered a case of this condition under the late Dr. Bristowe. It was found in this patient, if his memory served him right, that the topical application of iced water to the ligatured fingers induced a local destruction of the red blood-corpuscles, which, when readmitted to the general circulation, was followed by the passage of hæmoglobin in the urine. He had been unable to find any evidence of congenital syphilis in the present child. But, undoubtedly, the sister was syphilitic. He regretted the incompleteness of the notes of the case, owing to circumstances he had already mentioned, but he felt that it was a case that should be brought to the notice of the Society by reason of the early age at which the condition appeared, and by the fact that he was fortunately enabled to produce a specimen of its hæmoglobin-containing urine.

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22. A CASE OF SUPRA-RENAL SARCOMA.

By GEORGE CARPENTER, M.D.

Cyril A—, aged 2 years and 5 months, was admitted into the Evelina Hospital on August the 19th, 1901, with an enlarged abdomen. His maternal grandfather died of cancer of the stomach, and a female cousin on the maternal side, aged 11 years, of "cancer under the shoulder-blade." On the father's side there was a history of consumption, his mother and grandmother having died of it. Three weeks prior to his admission his stomach was found to be swollen.

On admission he was noticed to be a poorly-nourished, pallid child, with a pulse-rate of 120 to the minute, respirations twenty to the minute, and a temperature of 99·2° F. The *heart* and *lungs* were normal. His abdomen was distended, and on palpation the free edge of the liver could be felt about four fingers' breadth below the costal margin in the nipple line. Through the substance of the liver a tumour could be felt, and on tracing this tumour it appeared to be connected with the right loin, and occupied the right lumbar, hypochondriac, and a part of the epigastric regions. The hand on the abdominal wall could be insinuated between the tumour and the free edge of the liver, which was sharp and of natural consistence, for some little distance. The free surface of the liver was apparently quite healthy, and no lumps were detected in it, but it was thought that the tumour was attached to the under surface of the liver. In addition there was some irregular lumpiness in the umbilical, left lumbar, and hypogastric regions. There was no tumour springing from the left loin as on the right side. The spleen was not enlarged. The urine was healthy. A blood examination showed 60 per cent. of hæmoglobin, with a count of 3,500,000 red blood-corpuscles and 12,000 white blood-corpuscles per c.mm. A rectal exploration failed to yield any additional information, and an ophthalmoscopical examination gave negative results.

During the first week of his stay in hospital the temperature, by the rectum as well as the axilla, was for the most part subnormal. Then it rose a little above the normal, and on the 29th, when he was injected with five minims of tuberculin, it had been as high as 101·4° F. The injection was not followed by a reaction, and he was again injected on September the 6th with ten minims of tuberculin, and on September the 10th with fifteen minims of tuberculin, with negative results. Whilst under observation the abdomen became more swollen, and the abdominal lumps increased in size and hardness. He gradually became weaker and more anæmic, and died on September the 24th, having been in hospital thirty-six days. A few days prior to his decease a small tumour was found under the left sterno-mastoid muscle.

Autopsy.—On opening the abdomen, the liver was seen to be pushed upwards and over to the left by the tumour in the right half of the abdomen. The tumour was in the region of the right kidney and behind the peritoneum. Its surface was patchy or mottled and of a dark red colour, and the red staining (due to extravasation of blood) extended down into the iliac fossa and inwards to the greatly enlarged and infected mesenteric glands. Above, the tumour was firmly adherent to the liver, which was stretched in a thin layer over the tumour; but the liver was nowhere infiltrated, nor were any secondary deposits present in it. The right supra-renal body could not be found. The whole tumour, liver, and enlarged mesenteric glands were removed together, and weighed 3 lbs. 11 oz. (59 oz.). On longitudinal section a thin layer of liver was seen to be stretched over the upper extremity of the tumour, and separated from it by thickened peritoneum. Below this was the tumour—a dark red mass, grumous-looking and hæmorrhagic, and almost cystic in parts. At its lowest part was apparently normal right kidney substance, the amount remaining being about half the normal size of that kidney. Attached to the left of the lower part of the tumour was a large mass of infected mesenteric glands, some quite hard and almost gritty on section. The left kidney was very large, and found on section to be in a state of hydronephrosis.

Both ureters appeared normal, and a probe could be passed into the bladder along the terminal one and a half inches of each tube.

The spleen, pancreas, and left supra-renal capsule appeared normal.

The bronchial glands were considerably enlarged all along the aorta, and were secondarily infected.

Beneath the left sterno-mastoid muscle was a mass of lymphatic glands measuring about two inches by one inch, easily shelled out, but very soft and almost diffluent on section.

The right lung was much compressed by the tumour, which pushed up the liver and diaphragm. No secondary deposits were present in the lungs or any other internal organ. The brain was not examined, by request.

Microscopical Appearances.

(*a*) *Kidney and tumour.*—The tumour was a vascular small round-celled sarcoma, with a definite fibrous capsule separating it from the kidney substance. The kidney substance immediately adjacent showed great increase of the intertubular tissue, with atrophy of many glomeruli and tubules. Further away from the tumour the kidney tissue was almost normal.

(*b*) *Left kidney.*—This showed many dilated tubules; some glomeruli were cirrhotic and atrophied; an increase of intertubular tissue was found.

(*c*) *Infected gland in the portal fissure.*—This was found to be a round-celled deposit all through.

(*d*) *Infected mesenteric gland.*—This was similar to the portal gland. It showed a round-celled deposit and many hæmorrhages. (*Exhibited January the* 17*th,* 1902.)

Discussion.

The Chairman (**Mr. A. H. Tubby**) said the case was a very interesting one indeed. Unfortunately, sarcoma of the kidney had, for surgeons, a very melancholy interest. From time to time the condition presented very good opportunities for operating, but recurrence practically always took place. In a young child sarcoma of the kidney was very much like sarcoma of the femur in the matter of prognosis—very unfavourable. A point of considerable interest which had not been touched upon was as to the extent of growth which had been found in the renal veins or in the vena cava.

Mr. Clement Lucas agreed that cases of sarcoma of the kidney were most unfortunate ones. The growth extended so rapidly along the renal veins and lymphatics in the immediate neighbourhood that there was not much time for operation. Though the kidney would come out fairly well in those cases, the results were generally very unfavourable. He thought that Dr. Carpenter had acted wisely in not requesting a surgical colleague to operate in a case already so far advanced when diagnosed.

Dr. Cautley asked whether it was a case of renal sarcoma, or of extra-renal sarcoma pressing on the kidney. He thought the specimen showed that the kidney was not necessarily first involved, and suggested that the growth was primarily supra-renal.

Mr. Pernet asked whether any secondary sarcomatous deposits were noticed, clinically, in the skin.

Dr. George Carpenter, in reply, said he did not see any involvement of the renal veins or of the vena cava. The reason he did not seek operation was that on his first examination he thought it was tuberculous peritonitis, and when he arrived at the conclusion that it was not so it was obviously not a suitable case for surgical aid. As a rule renal sarcoma manifested itself as a single large mass springing from the loin, and there was in such cases resonance of the colon in front of the tumour. Nothing of the sort occurred in the present case; it was a diffused, irregular tumour occupying the greater part of the abdominal cavity, and suggestive of a general tuberculous matting of the intestines rather than abdominal sarcoma. Hence he injected tuberculin, but there was no reaction. Then he concluded it was a renal sarcoma with secondary implication of the abdominal glands. In reply to Dr. Cautley, his impression was that the tumour sprang from the supra-renal capsule, and invaded the kidney secondarily. Supra-renal tumours he regarded as uncommon. He had, however, in his experience, met with two other cases of supra-renal sarcoma with secondary involvement of the kidney. There was no involvement of the skin while the child was under observation, but there was a secondary deposit in the glands of the neck under the left sterno-mastoid muscle which was noticed a few days before its death.

23. A CASE OF TRAUMATIC CHOREA WITH MUSCULO-SPIRAL PARALYSIS COMPLICATING FRACTURE OF THE HUMERUS; REMARKABLE GROWTH OF HAIR OVER THE CUTANEOUS DISTRIBUTION OF THE NERVE.

By R. CLEMENT LUCAS, B.S.

L. T—, aged 11 years, was admitted into Guy's Hospital on October the 29th, 1901, for fracture of the upper third of the right humerus, caused by being run over by a cart. Twitching of the face was noticed when she was first brought to the hospital. An internal angular splint was put on the injured arm. About half an hour after admission the patient developed general chorea, which affected her left hand and arm, her legs, and slightly the head and neck. It was not noticed in the injured limb, which was steadied by a splint. On November the 19th the arm was put in a plaster-of-Paris splint; this was cut up on November the 26th, and the patient was discharged wearing it. There was a great improvement in the patient's condition as regards chorea when she left the hospital, and she says that it entirely disappeared two or three days after. She continued to wear the plaster splint till January the 6th, 1902, when the bone was found firmly united, but with a considerable amount of callus.

She was readmitted on January the 9th, for inability to extend the right hand. She carries her hand in a flexed position. The back of her right arm, from the site of the former fracture (about two inches below the head of humerus) to just above the elbow, is painful on pressure. The patient can extend the index finger and thumb, but only slightly extend the rest of the fingers. The hair on the back part of her right forearm is longer and more abundant than that on the left. This outgrowth of hair is very remarkable, being dark in colour, and about an inch in length. It also extends up on the outer side of the arm for its lower third, where it ends abruptly.

Remarks.—The patient is one of seven children born of healthy parents. There is no rheumatic history, nor had she recently had any illness, or suffered previously from chorea. No cardiac affection complicated the disease, and she developed no rheumatic nodules. Within half an hour of the injury twitching of the face was first noticed, and chorea became general (except in the broken arm) shortly after. Paralysis of the musculo-spiral nerve was not noticed when she entered the hospital, and it is believed to have been caused by the callus thrown out in consequence of the choreic movements preventing immobility at the seat of fracture.

It is proposed to cut down on the seat of fracture, and free the nerve from the callus pressing on it, if no improvement takes place as the result of electricity and massage. (*Exhibited January the* 17*th*, 1902.)

Discussion.

Mr. Pernet commented on the growth of hair on the injured arm, but said he could not explain it. He had seen the reverse occur after an injury to the head, namely, a patch of alopecia. But on the site of a burn he had seen an increased growth of hair. He had not seen hair develop on a case similar to the present.

Mr. Jaffrey asked whether the musculo-spiral nerve was injured at the time. He said he had seen an abnormal growth of hair in cases of hip disease, as no doubt other surgeons had also.

Dr. Frederick Taylor asked whether there was any other infectious disease which occurred comparatively recently, and whether it was certain that there were no choreic movements present before the accident. He said the relation of chorea to shock and fright was so obscure that it would be well to be assured on the two points he had mentioned. He asked in what sense Mr. Lucas used the term traumatic chorea. How did the traumatism operate—directly upon the central nervous system, or upon the musculo-spiral nerve, or by shock or emotion? A foreign writer had said that not only rheumatism, but scarlet fever and measles, and other infectious diseases might be causative antecedents of chorea.

Dr. George Carpenter asked if there had been any local irritant applied to the limb, which would account for the extra growth of hair. He called attention to cases of excision of the hip in older days, when, under the irritation caused by the carbolic spray, there was an unusual growth of hair in the neighbourhood of the wound and the parts exposed to the spray. He said that he had often noticed a growth of hair on the limbs of children who had long worn splints.

Mr. Clement Lucas, in reply, said one of the most interesting cases of growth of hair which had occurred under his observation was when he made a new eyelid for a child by turning a flap from the temple. From the fine hair in the skin of the temple which was transplanted long hair sprang, and produced an almost perfect eyelash, the vascular edge of the lid causing the increased growth. In answer to Dr. Taylor, he believed there was no exanthem or other disease preceding the accident. He said he was not a neurologist, but would be glad if any neurologist present would answer the question of the mode of action of the traumatism for him. He used the term because the condition almost immediately followed the injury, and the mother was emphatic about there being nothing of the kind present before. He thought it was open to question whether the implication of the musculo-spiral nerve produced the condition, whether it was the central nervous system, or whether it was the emotions. There was no head injury, and he could not throw any light on the case. Knowing that physicians usually attributed chorea to rheumatism, the latter was carefully inquired after, but with a negative result. His house-surgeon noticed that when the patient first came to the surgery there was twitching only on the left side of the face, and before she had been in hospital half an hour it had become generalised. He did not notice any choreic movement in the injured hand. It was in a splint, and he did not know whether there was any damage to the nerve at that time. Possibly, as in some cases he had brought before the Clinical Society within the last two years, the nerve may have got between the edges of the fracture. That was one of the causes of non-union. He drew attention to the fact that the increased growth of hair corresponded with the cutaneous distribution of the musculo-spiral ending abruptly above, at the junction of the lower with the middle third of the outer side of the arm, and covering the dorsal aspect of the forearm to the wrist.

Sequel by Mr. Clement Lucas (*September*, 1902).—It was determined before operating to try massage and electricity for the paralysed muscles. The contracted muscles were at the same time apposed by splints. The effect of this treatment was encouraging. At the same time the callus tended to decrease. At the end of three months the functions of the limb were completely restored. The abnormal growth of hair remained when last seen.

24. PARALYSIS TREATED BY MUSCLE GRAFTING, WITH NOTES OF TWELVE CASES.

By A. H. TUBBY, M.S.

For descriptive purposes the effects of an attack of infantile paralysis upon a limb may be divided into three degrees of severity. In the first place only one muscle or a group of muscles is either slightly affected, or, if apparently severely affected, yet eventually undergoes a very considerable recovery ; in the second group may be placed those cases in which one or more muscles of a group do not recover, and remain functionless ; and the third group comprises those severe cases in which all the groups of muscles around a joint are hopelessly paralysed, and the joint is flail-like.

The first class of cases includes those treated by simple tenotomies, massage, galvanism, and the use of appliances. The second group may either be treated in the same way, or, to much better advantage, by tendon grafting ; while to the third group arthrodesis is particularly applicable.

Now as to the *rationale* of tendon grafting : the condition of the part is such that, so far as function is concerned, extensive loss has taken place in one part, while another part remains intact, so it therefore follows there is a loss of balance ; hence the deformity.

The objects of tendon grafting are twofold : first of all, to utilise what has now become ill-directed voluntary movement ; and secondly, by so doing to restore the balance of muscle in the affected part. But it will be evident that the joint can never be made a fully strong one, since the amount of voluntary action of muscles at the disposal of the surgeon has from the outset been diminished. Yet it must be conceded that a well-balanced weak joint is better than an ill-balanced weak joint. Still too much must not be expected from tendon grafting, for there are many difficulties to be overcome. There are, of course, anatomical and surgical limitations in the transference of tendons from one side of a joint to another. It is also difficult to regulate to a nicety the amount of power transferred, and it occasionally happens that, some time after the operation, an apparently functionless muscle, when it is no longer unduly stretched by over-action of its opponents, will recover somewhat and disturb the balance which has been obtained. Yet the method presents great possibilities, and the results are often gratifying, being far superior to the methods hitherto employed of tenotomy and the use of apparatus; because, in place of passive retention of the foot by apparatus in a more or less normal position, voluntary power—already present and running to waste—is now directed to overcoming the deformity.

There are certain points which need close attention in tendon grafting.

1. All faulty positions of the foot, especially those due to secondary contraction of fasciæ, such as pes cavus, will require attention.

2. The electrical reactions of the muscles should be ascertained previously.

3. For mechanical reasons it is advisable to select the reinforcing tendon from a muscle whose line of action is as nearly parallel as possible to that of the muscle to be reinforced. For instance, in treating paralytic valgus it is better to graft a strip of the tendo Achillis into the tibialis posticus than to bring the tendon of the peroneus longus across the front of the ankle and graft it into the tibialis anticus.

4. It is scarcely necessary to insist upon absolute asepsis in the operation.

5. In grafting one tendon into another the foot should be held in the position it is desired to obtain, and when the tendons have been sutured, both the reinforcing and receiving tendon should be quite tense.

6. After the operation the part should be kept at rest for at least six weeks in the correct position, to allow of firm union taking place.

7. For a long period after the operation every effort should be made to develop the reinforcing muscle by massage and the application of electricity.

8. A careful watch should be kept upon the foot from time to time to see that the balance which has been secured is maintained.

The possibilities of tendon grafting are great, and allow much scope for ingenuity, but each case must be carefully considered on its merits, and the steps of the operation planned beforehand.

Of the cases which I have shown to-night four have been done for calcaneo-valgus, two for equino-valgus, and two for equino-varus, one for valgus, and one for paralysis of extensor cruris, and one for infantile hemiplegia. The results in calcaneo-valgus are better than those in the other groups of cases, because in the peroneus longus we have a tendon which for a long distance runs parallel with the tendo Achillis, and is easily inserted into it without much disturbance of the part, while the division of the peroneus longus tendon diminishes the excessive action of the evertors of the foot.

In equino-varus a good result can frequently be obtained. By splitting the tendo Achillis for a long distance upwards, and freeing the lower end of the inner segment and attaching it to the tibialis posticus, a powerful invertor is formed. If at the same time the remaining segment be divided transversely the equinus portion of the deformity is rectified.

As to the material to be used for joining one tendon to another, I find that none answers so well as fine Chinese silk boiled for half an hour, and then placed in a solution of 1 in 1000 biniodide of mercury. I have experienced no difficulty with the silk afterwards in any of my cases.

Arthrodesis is adapted, as a rule, only to the third group of cases, where the joint is flail-like from complete paralysis of the muscles around it, and it finds its best use at the ankle.

The object of the operation is to produce a firm ankylosis of the bony surfaces, but it is curious to note how extremely difficult this is to obtain, and many measures have been tried. I have not succeeded in satisfying myself that any of the cases which I have operated upon have been followed by bony ankylosis, but in several there has been very close fibrous union of the joint surfaces, which has answered the purpose quite as well. It is evident that, in a case with a flail-like ankle, if the joint surfaces can be made firm, heavy supporting apparatus can be dispensed with, and the patient will be much benefited by the operation.

With regard to the boy T. P—, whom I have shown to-night, his case is of an entirely different character from those spoken of previously. He has suffered from infantile hemiplegia, and has the deformity typical of that disease both in the upper and lower extremities. The left elbow is flexed, the forearm pronated, the wrist and fingers flexed, and the thumb is adducted and bent into the palm. He also has talipes equinus of the left foot. In the case more particularly of the upper extremity there are two factors to be taken into consideration in applying any remedial measures, namely, contraction (*a*) of the muscles, and (*b*) of the ligaments. By the method I adopted in his case, as well as in other cases, not only can the contraction of the muscles be overcome, but power, which under the circumstances of the disease is viciously directed, can be transferred in such a way as to make it an agent for useful motion. The contraction of the ligaments can subsequently be overcome by passive movements, once the muscular contraction has been remedied.

The operation which I have practised is described in the 'British Medical Journal' of 1898.

After the operation the improvement is slow, and great persistence is necessary, both with massage and with passive movement, and the patient should be encouraged to use his arm. To induce him to do so I frequently tied up the sound arm.

There are doubtless improvements to be made in the methods of operation, but the results I have obtained so far are encouraging.

Cases of Talipes Calcaneo-valgus treated by Tendon Grafting.

CASE 1.—I. F—, aged 8 years, was admitted into Westminster Hospital on October the 19th, 1900, suffering from paralytic talipes calcaneo-valgus of the left foot, which had come on after a paralytic attack when she was two years of age. She had received no treatment for the condition until she came under my care. When the limb was passively raised from the ground the front part of the foot hung helplessly, pointed and everted, the sole was deeply concave, the heel was prominent, and had the typical globular appearance of these cases, and the plantar fascia was contracted. The latter was divided, and the sole of the foot unfolded, a process which took about four weeks. The muscles having been carefully tested, it was found that there was some power left in the tibialis posticus, and normal power in the peroneus longus. It was therefore decided to graft the tendons of the peroneus longus and the tibialis posticus into the tendo Achillis, which was accordingly done. The condition of the foot and the result of the operation are well shown in the illustration on page 82 (*vide* Figs. 5, 6, and 7). The child can voluntarily raise the heel from the ground; the foot is of comely shape, and she walks with ease.

CASE 2.—A second case of calcaneo-valgus was that of A. S—, aged 6 years, who was admitted into the National Orthopædic Hospital on June the 23rd, 1898. It was noted that the peronei muscles acted well, but the calf muscles failed to raise the heel at all. On July the 1st the peronei tendons were grafted into the tendo Achillis behind the external malleolus. On August the 10th it was noted that there was good power of voluntary extension of the foot, and when it was made the peronei muscles

To Illustrate Case 1.

Fig. 5.

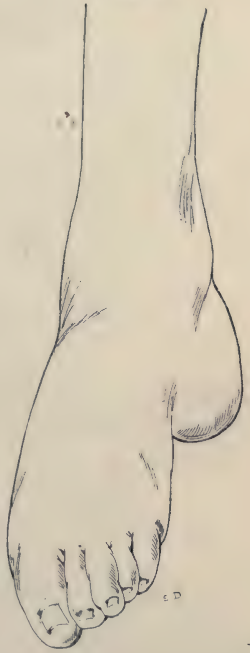


Fig. 5.—Before Treatment.

Showing the paralytic valgoid condition of the foot.

Fig. 6.

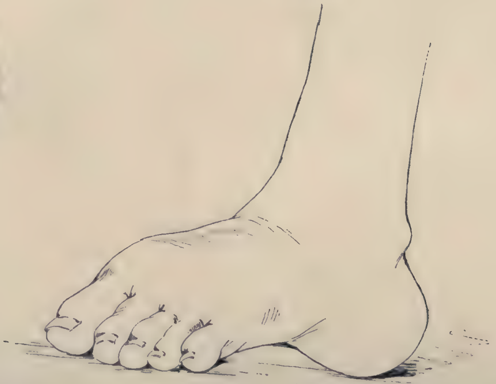


Fig. 6.—Before Treatment.

Same foot as in Fig. 5, showing the globular heel due to the talipes calcaneus.

Fig. 7.

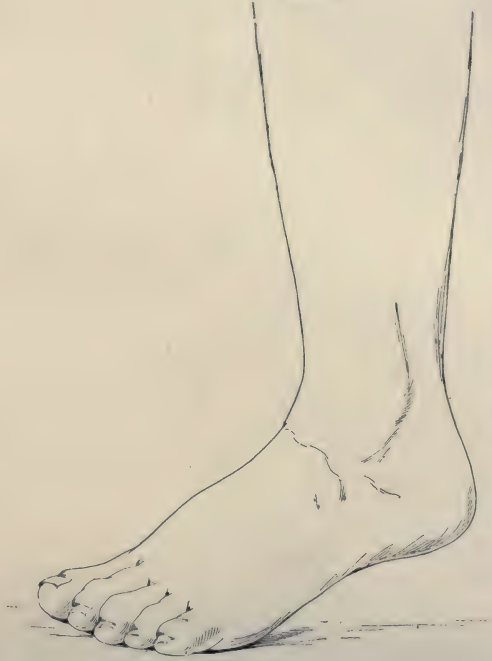


Fig. 7.—Restored.

Showing the restored power of raising the heel and inverting the foot effected by muscle grafting.

could be felt to harden, and the lower portion of the tendo Achillis became tense. There was no valgus, and the foot was in excellent position. He could lift the heel off the ground, and raise himself on his toes. For a time he wore a light steel apparatus, so as to control the valgus; but this has since been discarded, and the improvement is maintained.

TO ILLUSTRATE CASE 3.

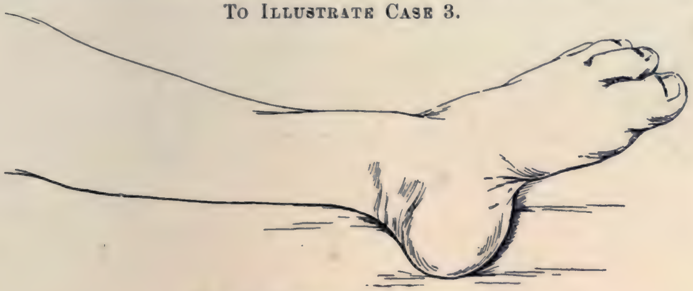


FIG. 8.—BEFORE TREATMENT.
Case of paralytic talipes calcaneo-valgus, showing the dropped heel and everted foot.

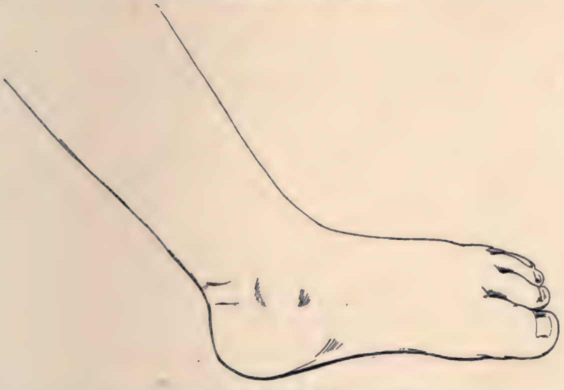


FIG. 9.—AFTER TREATMENT.
The result of grafting the peroneus longus tendon on to the tendo Achillis.

Case of Calcaneo-valgus treated by grafting the Peroneus Longus Tendon into the Tendo Achillis.

CASE 3.—W. C—, aged 3 years, was admitted into the National Orthopædic Hospital on September the 19th, 1899, suffering from infantile paralysis of the left leg. When about a year old, and after he had commenced to walk, he had a convulsion. It was noted on admission that the muscles of the left leg were generally wasted, and the plantar flexors and invertors were paralysed, while the dorsiflexors and evertors retained a moderate degree of strength. The right foot was not much affected, but the sole of the foot was somewhat more hollow than natural, and the balls of the toes were dropped. On October the 28th, 1899, the left peroneus longus tendon was grafted into the tendo Achillis, and the patient was discharged from the hospital on February the 22nd, 1900. He was able to raise himself on his toes, but some talipes valgus persisted, which was controlled by a valgus pad and T-strap, and this is his present condition.

Case of Paralytic Talipes Valgus treated by grafting part of the Extensor Communis Digitorum Tendon into the Tibialis Anticus Tendon.

Case 4.—F. L—, aged 4 years, was admitted into the National Orthopædic Hospital on February the 20th, 1900. The history was that a year before admission, when he woke up one morning, it was found that he could not walk, although he had gone to bed quite well the night before. There was much wasting of the left leg, and the knee-jerk on the left side was not obtainable. The foot was everted, and there was good power in the peronei and the extensor communis digitorum muscles. When the foot was brought to the middle line he was able to invert it slightly by the action of the tibialis posticus muscle, although the tibialis anticus appeared to be paralysed. The limb was half an inch short. On February the 22nd a transverse incision was made across the front of the ankle-joint, the extensor communis tendon found and split longitudinally, and, its outer half having been divided, the proximal portion of this section was grafted on to the tibialis anticus tendon. It was not deemed

To Illustrate Case 4.

Fig. 10.

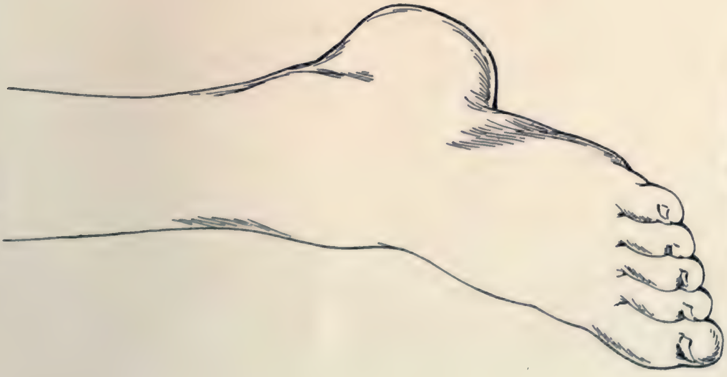
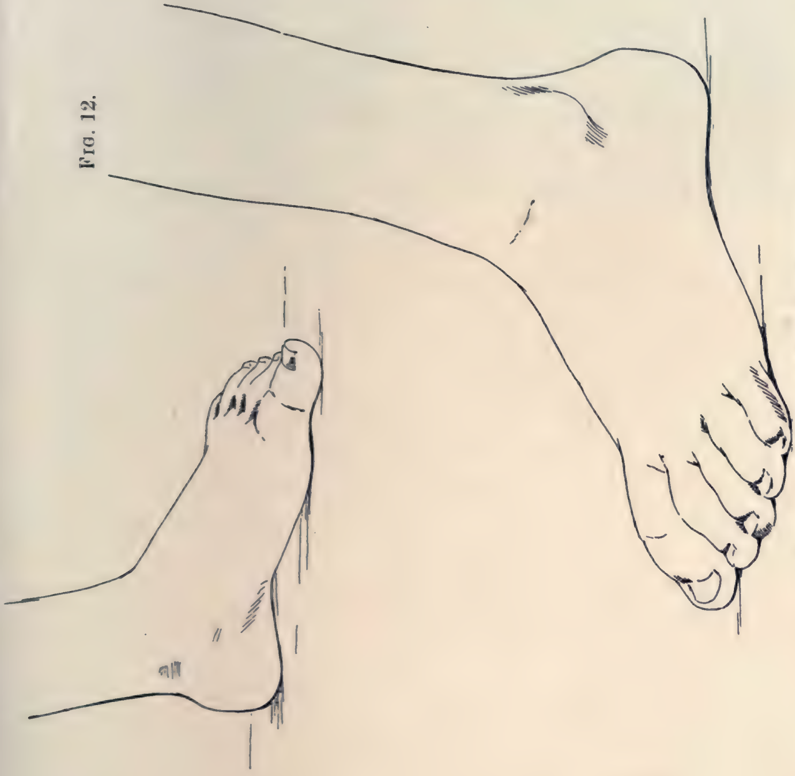


Fig. 10.—Before Treatment.
A case of paralytic valgus.

Fig. 11.

Fig. 12.



Figs. 11 and 12.—After Treatment.
The same foot, after treatment by grafting the inner half of the extensor communis digitorum tendon on to the tendon of the paralysed tibialis anticus muscle.

advisable to graft the peronei tendons on to the tendo Achillis, as the amount of calcaneus was not great. The effect of the operation was that he could raise the inner border of the foot, and much of the valgus was overcome. When seen on July the 18th, 1901, the result was very good indeed, and fully justified the operation. The calcaneus had disappeared, as the result of persistent massage and faradism of the calf muscles.

Case of Talipes Equino-valgus treated by Tendon Grafting.

Case 5.—C. L—, aged 6½ years, was admitted into the National Orthopædic Hospital on August the 29th, 1898, suffering from infantile paralysis of the left foot, which was in a position of equino-valgus. The tibialis anticus and posticus muscles were paralysed, while the extensor proprius pollicis and inner portion of the extensor communis digitorum muscles were affected in a less degree, but the outer portion of the latter seemed to act well, and so too did the peronei. The tendo Achillis was somewhat tight, and the angle of dorsiflexion was just under ninety degrees. The sole of the foot was more concave than usual.

On October the 12th, some weeks after section of the plantar fascia, an incision, five inches long, was made along the inner and posterior aspect of the leg, and the tendo Achillis was split along its whole length; the inner half was severed below the ankle, and brought round and stitched to the tibialis posticus tendon. The remainder of the tendo Achillis was divided, as well as the peronei tendons. All went well, and when the boy left the hospital in January, 1899, his foot was in a capital position, but since then slight relapse has taken place, and there is now some valgus present. It is a matter of regret that a portion of the peronei tendons was not exsected; or it would probably have been better to carry round the peroneus longus tendon into that of the flexor communis digitorum, as well as inserting the inner half of the tendo Achillis into the tendon of the tibialis posticus muscle.

Cases of Talipes Equino-varus treated by Tendon Grafting.

Case 6.—C. C—, aged 2¼ years, was admitted into the Evelina Hospital on July the 6th, 1898. He appears to have had an attack of infantile paralysis at the age of nine months. He was a healthy-looking boy, but the right leg was partially paralysed; this was especially so with the extensor muscles of the toes. The foot was pointed and inverted, and rested on the external malleolus.

On July the 22nd a longitudinal incision, from three to four inches in length, was made at the posterior and outer aspect of the leg, and the tendo Achillis and peroneus longus tendon were exposed. The tendo Achillis was then split longitudinally for its whole length, and the gastrocnemius tendon as far as the junction of its two heads. The outer part of the tendo Achillis was then severed transversely about an inch and a half above the ankle, and so, too, was the peroneus longus tendon. The proximal part of the tendo Achillis and the distal part of the peroneus longus tendon were then firmly quilted together; the remaining inner portion of the tendo Achillis was then severed, so as to correct the equinus. For six weeks after the healing of the wound massage and faradism were applied; it was evident that the everting movement of the foot had been regained. The present condition is that the boy is able to walk about comfortably without any instrumental assistance, the only drawback being that the leg is one inch shorter than its fellow.

Case 7.—D. O'G—, aged 2½ years, was seen on December the 6th, 1898, with infantile paralysis affecting the left leg. The foot was in a position of equino-varus, and the muscles affected were the same as in the preceding case. The same procedure was carried out, but the outer portion of the gastrocnemius tendon and the outer portion of the tendo Achillis were freely detached. For two years the foot remained in good position, but the extensor communis digitorum muscle recovered its power almost completely, so that the two outer tendons became tense, and the foot acquired some valgus, which will have to be corrected by section of the affected tendons. It must be remembered that long after an attack of paralysis the affected muscles continue to recover, and may therefore upset the balance which has been obtained previously by tendon grafting. Particularly so is this the case with the extensor communis digitorum muscle.

Case of Talipes Equino-valgus treated by Tendon Grafting.

Case 8.—B. F—, aged 9 years, was admitted into the National Orthopædic Hospital on October the 5th, 1899, with a condition of talipes equino-valgus of both feet, which was undoubtedly of paralytic origin, the muscles of the left leg being particularly wasted. No history was obtainable of the onset of the paralytic condition, but all the symptoms were those of infantile paralysis. Both feet were dropped owing to partial loss of power in the extensor muscles of the feet, and the valgoid position appeared to be due to paralysis of the tibialis posticus and flexor longus pollicis muscles.

On February the 1st the tendines Achillis were divided and also the plantar fasciæ, and on March the 22nd the outer two tendons of the left extensor communis digitorum were implanted in the tibialis posticus tendon. The child walked well with the aid of apparatus, but the left foot has shown some tendency to relapse into the valgoid position.

Case of Paralytic Talipes Calcaneo-valgus treated by Tendon Grafting.

Case 9.—B. K—, aged 6 years, was admitted, on October the 8th, 1901, into the National Orthopædic Hospital, suffering from paralytic talipes equinus. The left lower limb was wasted, and was shorter than its fellow by one inch. The flexor and extensor muscles of the knee were weak, but the sartorius muscle was active; so too were the extensor muscles of the toes. The foot itself was in a position of talipes equinus, and the sole was hollow.

On October the 17th a section of the plantar fascia was made, and on October the 24th muscle grafting was undertaken. A long incision was made over the inner border of the tendo Achillis, which was isolated, and the peronei tendons were exposed. Both the peronei tendons were divided, and the peroneus longus tendon was inserted into the tendo Achillis. A transverse incision was then made across the front of the ankle, and the outer two tendons of the extensor communis digitorum were inserted into the tibialis anticus tendon in such a way as to bring the foot into a position of varus. The wound healed kindly, and the leg is now being galvanised. There is a fair amount of power in raising the heel off the ground, and the foot is held fairly well to the front.

Case of grafting the Semi-membranous Tendon into the Tendon of a Paralysed Extensor Cruris.

Case 10.—L. O—, aged 13 years, was admitted into the National Orthopædic Hospital on September the 5th, 1901, suffering from paralytic talipes equino-varus of the left foot, and complete loss of power of extension in the leg. The position of the parts was as follows:—The limb hung at a right angle at the knee, and she could not extend it. The heel was slightly raised, and the fore-part of the foot was adducted and inverted. The plantar fascia was tense, and there was contraction of the sole of the foot.

On October the 17th a curved incision was made across the upper margin of the patella and along the inner surface of the thigh, extending about six inches. Search was made for the sartorius muscle, but it could not be found, so a large slip of the semimembranosus muscle was isolated for six inches, separated at its lower end, and attached to the front of the rectus femoris tendon with silkworm-gut sutures.

The after history of the case was uneventful, and the patient, although now unable to raise the limb off the bed with the knee extended, yet offers considerable resistance to the hand when an attempt is made to forcibly flex the knee, so that the case promises well.

Case of Infantile Paralysis treated by Arthrodesis of the Ankle-joint.

Case 11.—A. S—, aged 13 years, was admitted into the National Orthopædic Hospital on February the 21st, 1901, with severe infantile paralysis of the left leg and foot. She is stated to have begun to walk at the usual age, but when she was rather more than two years old she had a convulsion, and next morning the left leg was found to have lost power. Since then she has been unable to walk without the aid of crutches. On admission the left leg below the knee was seen to be extensively wasted and the muscles entirely paralysed. The left foot hung flail-like. There was also considerable loss of power in the muscles of the left thigh, and the knee-joint was loose, and could be hyper-extended with ease.

The flail-like condition of the ankle-joint was treated by arthrodesis. An anterior incision was made from the tip of the external to the tip of the internal malleolus across the front of the joint, and each tendon was isolated and ligatures threaded through, one above and one below the site of intended division of the tendon. The tendons were then divided between the ligatures and the ankle-joint opened. All the articular cartilage of the joint was then carefully removed, and the bones stippled with a gouge. The tendons were then reunited and the wound closed. Firm and close fibrous ankylosis resulted, and the foot is now of use to her as a means of support. The knee, however, continues to be controlled by steel apparatus.

Case of Spastic Paralysis of an Arm treated by Tendon Grafting.

Case 12.—T. P—, aged 7 years, was admitted into Westminster Hospital on May the 25th, 1901, suffering from spastic paralysis of the left arm and leg. The forearm and hand were in the typical position of these cases, and the foot was in a position of equinus.

On June the 1st the patient was anæsthetised and an incision was made on the front of the left forearm about three inches long, and just inside the anterior edge of the radius. The radial vessels and nerves were exposed and hooked aside, and the insertion of the pronator radii teres muscle carefully defined. Its tendon was separated from the bone. The interosseous membrane was then pierced and the tendon

TO ILLUSTRATE CASE 12.

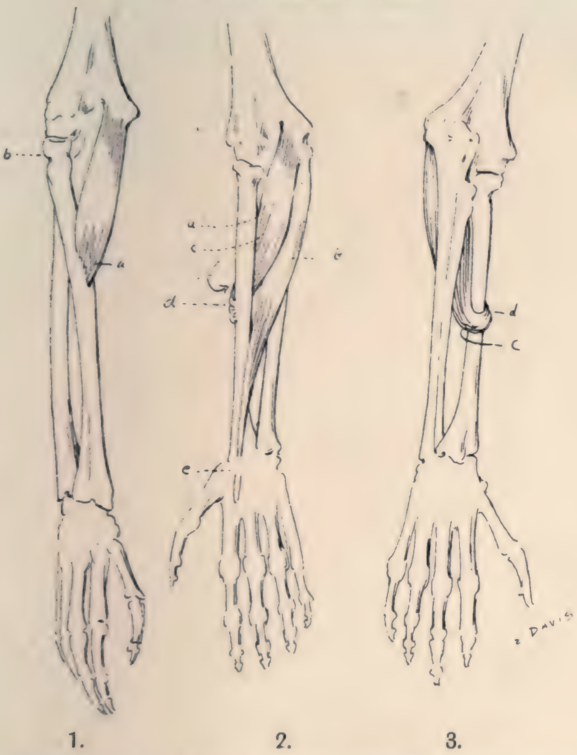


FIG. 13.—To illustrate the method of transforming the pronator radii teres muscle into a supinator. *a.* Pronator radii teres, in 1 passing in its natural course to its insertion on the radius, and in 2 its tendon has been detached, and has been passed *behind* the radius to its outer side. *c* is the opening in the interosseous membrane through which the tendon is passed, and it is re-inserted at *d.* At *e* the tendon of the flexor carpi radialis muscle has been divided. 3 is a posterior view of 2, but in 3 the pronator radii teres muscle is drawn too low down on the radius.

To Illustrate Case 12.

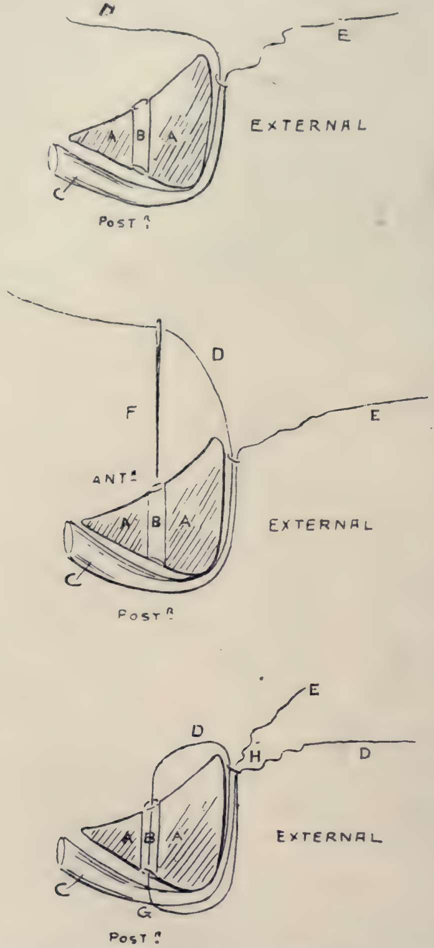


Fig. 14.—To show the method of re-inserting the pronator radii teres tendon into the radius, after the tendon has been passed behind the radius. A, A, section of the radius. B, a hole drilled through the bone from before backwards. C, the tendon of the pronator radii teres muscle. D and E, ends of silk thread passed through the detached tendon. F, needle threaded on D and passing through the hole B in the radius in the second figure, and through the tendon of the pronator radii teres muscle at G in the third figure. G, position where the free end of the thread D is passed through the tendon as it lies behind the radius. H, spot where the thread D is finally knotted to E, and the tendon is bound down to the roughened outer surface of the radius at its new attachment.

drawn through it. With a drill the radius was pierced, and the tendon having been drawn round the back of the radius was fixed in its new position by a ligature passing through the hole in the radius (Fig. 14). The flexor carpi radialis tendon was also divided and the wound closed. The boy has at this date (January, 1902) very fair power of supination, whereas before the operation he had none whatever. (*Read January the* 17*th*, 1902.)

25. A CASE OF TRAUMATIC DISLOCATION OF THE HIP-JOINT OF SIX WEEKS' STANDING; REDUCTION.

By A. H. TUBBY, M.S.

Edith B—, aged 5½ years, was admitted into the Evelina Hospital on January the 6th, 1902.

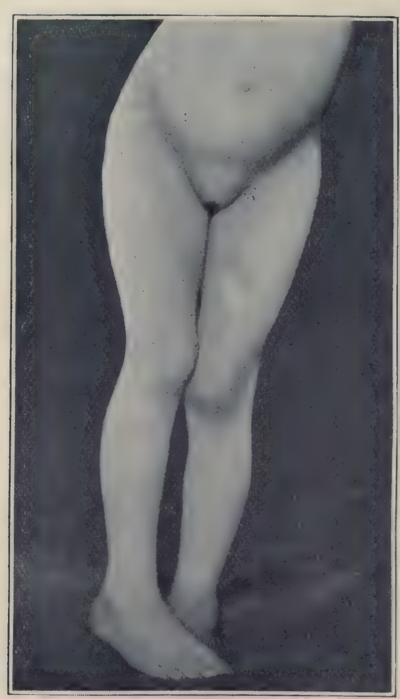
History.—Six weeks ago the child was playing on a high chair and fell out of it, and could not get up. What her attitude at the time of the accident was cannot be said, as she only had some small children with her. When first seen by an adult it was noticed that the right leg was underneath her. The mother was sent for and the child was taken to a doctor. The mother expressed the opinion that the small bone of the knee was hurt because the child had pain in the knee, and she was treated for a sprain. A week ago the mother felt what she thought was the bone out of place, so she compared it with the other side and found it very different. The patient could not walk.

On examination the attitude is very characteristic. She lies with marked adduction of the right leg, also there is obvious shortening (found on measurement to be two inches). There is no rigidity such as is found in tuberculous disease; on the contrary, there is abnormal mobility.

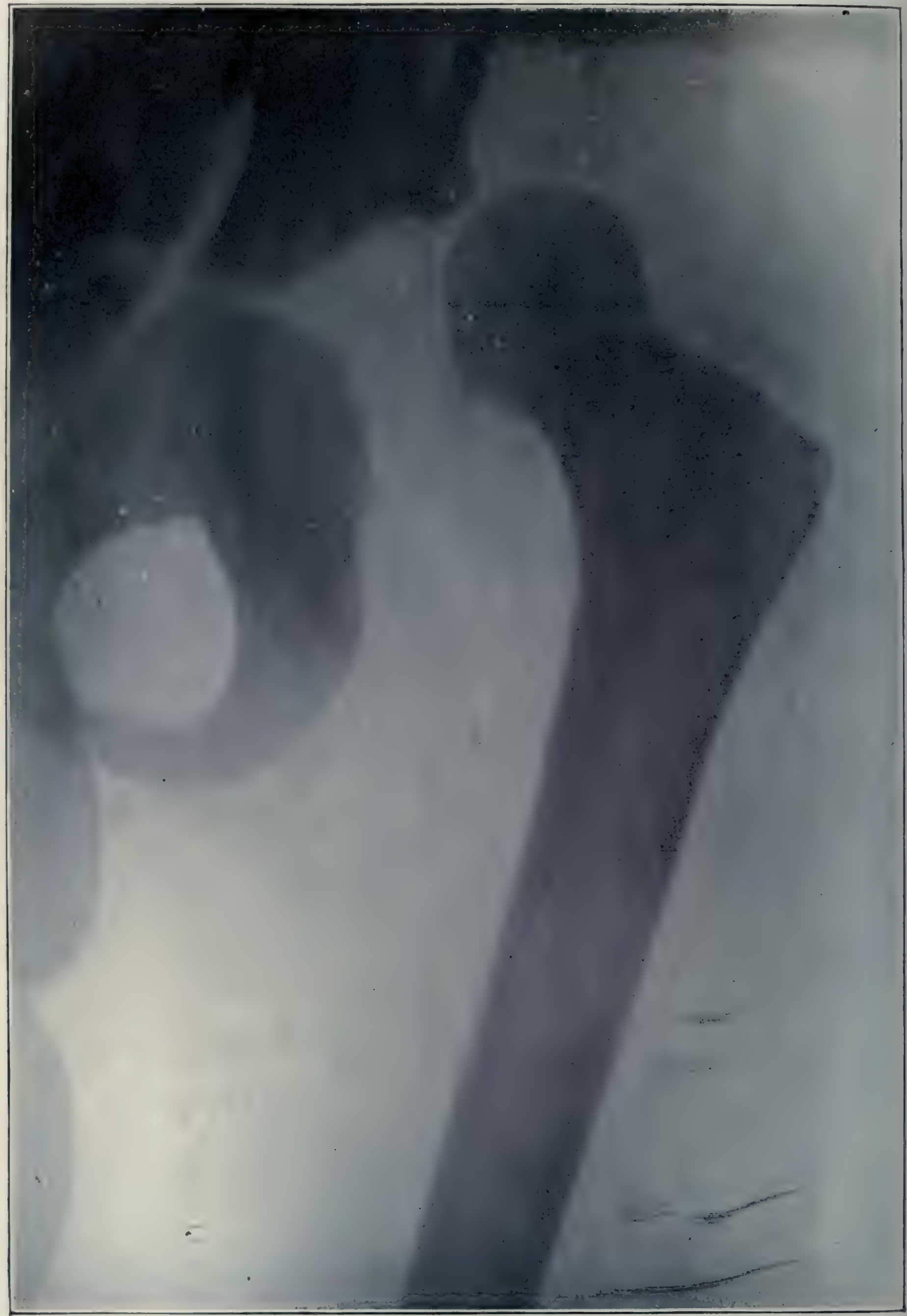
On feeling over the front of the joint, and comparing the two sides, it is noted that on the affected side there is absence of the normal feeling of resistance; the great trochanter is well above Nélaton's line, and the head of the femur can be felt above the dorsum ilii.

The hip was X-rayed, the photograph showing a dorsal dislocation of the head of the femur, the acetabulum being quite empty (*vide* Plate I).

FIG. 15.



On Saturday, January the 11th, chloroform was administered, and by extension, flexion, abduction, circumduction, and extension, the dislocation was reduced. The head of the femur went back into the acetabulum fairly easily, but probably owing to a large tear in the joint capsule it was



Mr. A. H. Tubby's Case of Traumatic Dislocation of the Hip-joint in a child aged 5½ years.

Adlard & Son, Imp.

very liable to slip out again. The leg was put up in splints, anterior and external, of soft material. The length of the legs was then found to be equal, the folds of the buttock on the affected side normal, and the right great trochanter in normal position.

(*Exhibited January the* 17*th*, 1902.)

Discussion.

Mr. Clement Lucas said the suggestions made by Mr. Tubby were extremely valuable, and likely to produce very great improvement in cases of paralysis which occurred in the lower extremity. The difficulty of producing ankylosis was only what one would expect in paralysed limbs. There was no tendency to form strong adhesions; and even in healthy children there was never bony ankylosis after excision of joints. Such cases as had been shown indicated that it was an advantage if a certain limited amount of movement remained. The same argument applied in the case of the last child. If the condition had been present in an adult there would have been greater difficulty in reducing the dislocation in five weeks. Not only were the surfaces of the joint more shallow, but the tendency to formation of fibrous adhesions was very much less in a child than in an adult.

Dr. A. E. Sansom also praised Mr. Tubby for his contribution, which dealt with a recent departure. It was a splendid combination of the mind and skill of the surgeon with the mind and thought of the physician. One of the things which struck him in the case was the possibility of surgical interference in infantile hemiplegia, which was in his experience not infrequently mistaken for infantile paralysis, a totally different disease. Infantile paralysis was one thing, and infantile hemiplegia was another. He had seen cases of infantile hemiplegia which had arrived at a considerable age, with very great deformity, and he had observed strange things happen in such cases, *viz.*, an acute illness grafted on the chronic. In one such, influenza occurred in a case of infantile hemiplegia, and a fine tremor of all the affected muscles arose while it was under observation. Cheyne-Stokes respiration also occurred in this case, nevertheless recovery took place. It was rare that recovery followed Cheyne-Stokes respiration, except in an acute disease. That case was acute, but it was grafted upon an old trouble. Little drawbacks occur to the physician's mind in connection with such cases as Mr. Tubby had brought forward, namely, that what had occurred once might possibly happen again; thus, in some of the cases which had been rectified so much by the surgeon, there might be a repetition of the neuro-vascular mischief.

26. A CASE OF MYOSITIS OSSIFICANS.

By GEORGE CARPENTER, M.D.,

AND

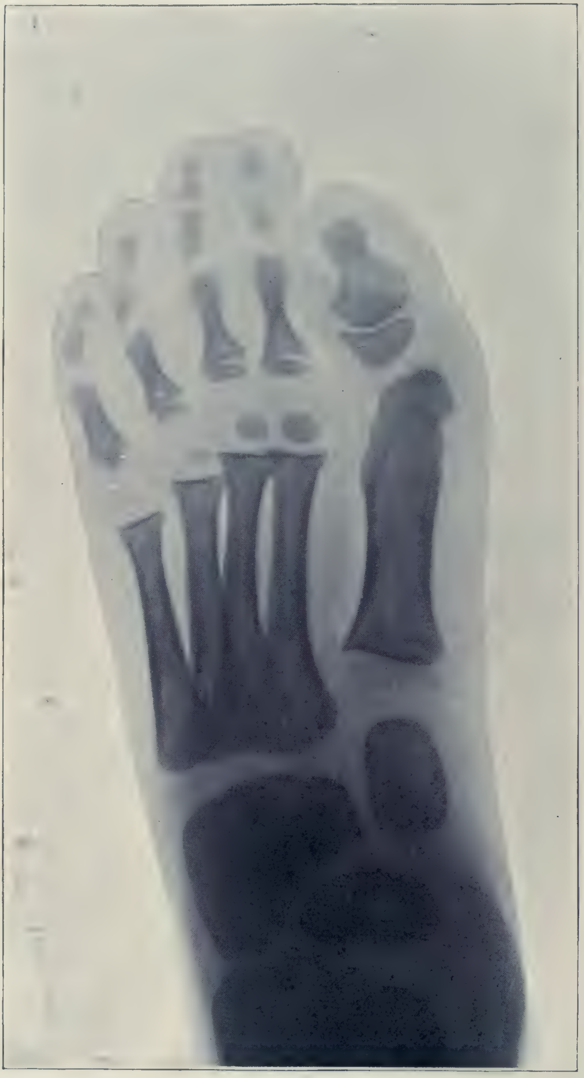
WALTER EDMUNDS, M.C.

Edith W—, aged 4 years, was admitted into the Evelina Hospital on August the 23rd, 1901, having previously been an out-patient for two or three weeks.

Family history.—There is no history of myositis ossificans in the family. Her father and mother are both alive and quite well; there are eight other children in the family, and all are healthy. Her mother had experienced neither miscarriages nor stillbirths, and not one of her children had "snuffles" during infancy. There is no history of tuberculosis in either the father's or the mother's family.

Previous history.—The patient has been a healthy child, but is nervous, and sometimes after a cold the glands in her neck become swollen.

Present illness and condition.—Six months ago she had both tonsils removed, and a fortnight later a hard and tender lump about the size of an egg appeared on the right side of the neck. The swelling, which appears to have been an inflamed gland, disappeared in a fortnight, and a similar swelling arose a fortnight later on the left side, which also quickly disappeared. Swellings subsequently appeared in the left breast (pectoral muscle), the region of the left scapula, and in the lumbar region. In the left sterno-mastoid muscle a hard spicule of bone can be felt, and the muscles of the neck on the left side are infiltrated and hard; this induration extends backwards to the middle line behind; the skin over this infiltrated area is movable. The patient holds her head stiffly. In the middle line of the neck in front there is a bony infiltration of the genio-hyoid muscles, which produces a pro-



Dr. George Carpenter's and Mr. Walter Edmunds's Case of Myositis ossificans, in a child aged 4 years, showing Microdactyly.

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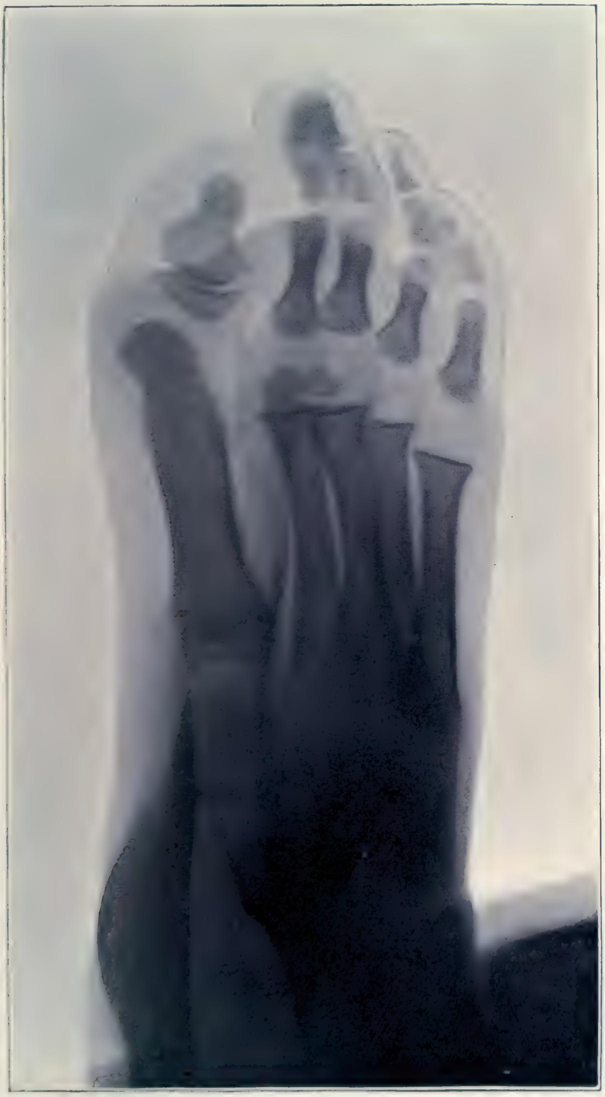

Dr. George Carpenter's and Mr. Walter Edmunds's Case of Myositis ossificans,
in a child aged 4 years, showing Microdactyly.

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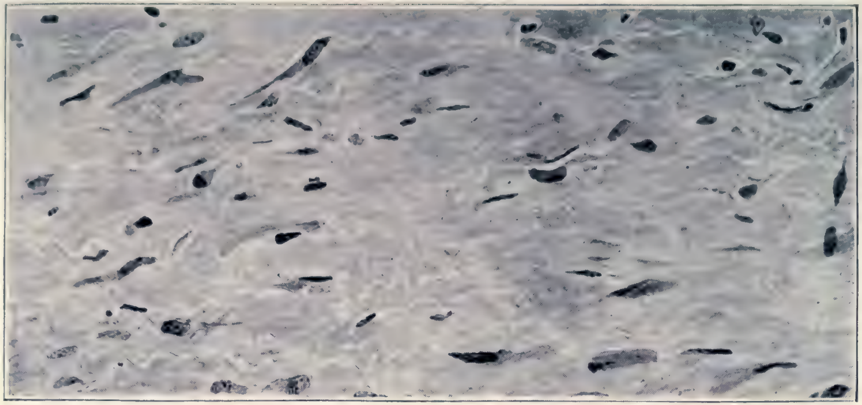

Dr. George Carpenter's and Mr. Walter Edmunds's Case of Myositis ossificans in a child aged 4 years.

Microscopical section showing fibro-cellular tissue; taken from a muscle which revealed, on exploration, grey infiltration.

Adlard & Son, Imp.

minence the size of a pea. In the right sterno-hyoid muscle there is also a bony infiltration. In the right arm there is a bony infiltration in the coraco-brachialis muscle. In the left pectoralis major muscle there is a distinct bony plate. Movements of the right upper arm are limited on account of the bony growth. There is a brawny infiltration in the erector spinæ region, the skin being to some extent bound down to the underlying muscle. There seems to be fusion of the two phalanges of the great toe, a condition recognised as occurring in these cases (see radiograms, Plates II and III). The internal organs, the knee-jerks, superficial and deep reflexes, and the optic discs are normal.

On September the 3rd, 1901, an incision was made in the lower part of the back over this brawny area. The skin seemed normal, but there was in the subcutaneous tissue and underlying muscular region a grey infiltration. Portions of this were removed for microscopical examination. No normal muscle-fibres were seen, although the incision was purposely enlarged to find them. The removed tissue showed nothing but fibro-cellular infiltration (*vide* Plate IV). No micro-organisms were detected.

A very interesting case of this disease by Herringham is recorded in the thirty-second volume of 'The Clinical Society's Transactions.'

The patient is seen again to-day, January the 17th, 1902, after a long interval. The disease seems to have advanced considerably. The various bone centres previously mentioned are enlarged, and there are now others, one on each side, lumps the size of a chestnut on the ribs near the angles of the scapulæ.

(*Exhibited January the* 17*th*, 1902.)

Discussion.

The Chairman (Mr. A. H. Tubby) said it was a very chronic case, but unfortunately no cause for the disease was known. Dr. Nabarro had made investigations in such cases, but had found no clue. At Westminster Hospital he (Mr. Tubby) had a case in a man aged 25 years, whose back was bowed. What determined the ossification of the muscles it was very difficult to say.

Postscript by Dr. George Carpenter and Mr. Walter Edmunds (*October*, 1902).—This patient was seen again on September the 30th, 1902, and the following notes were made on her condition at that time:

The lump of bone under the chin is larger. On the left scapula posteriorly there is a bony mass the size of a walnut, with separate bosses on its surface, which is immovably attached to that bone.

On the left flank there is a ridge of hard bone running downwards and inwards towards the spine, from a point external to the angle of the scapula to the level of the twelfth dorsal vertebra.

On the right scapula at its lower angle are bosses similar to those described on the left, but they are not so prominent. On its vertebral border there is also a more diffused mass of new bone.

The spines of the six lower dorsal vertebræ are affected with bony growth. There is also a bony mass on the left side of this part of the spinal column, and the spine is, in consequence, very rigid.

In the left pectoralis major muscle there is a mass of bone which prevents the arm being raised above the horizontal level.

On the right side there is a bony mass in the pectoralis major muscle, and bony growths can be felt in the coraco-brachialis and sterno-mastoid muscles.

The child has recently had measles, and has lost flesh in consequence.

Owing to the rigidity of her spine she cannot rise unaided when in the lying-down position.

27. A CASE OF CHRONIC INTERSTITIAL NEPHRITIS IN A BOY AGED SEVEN YEARS.

By W. A. MILLIGAN, M.B.

(Introduced.)

The patient, a boy aged 7 years, was first brought for advice in December, 1899, the parental complaint being that he was getting very thin, very white, and frequently complained of headaches. On examination he was found to have symptoms corresponding to those of chronic interstitial nephritis. He was treated for this disease till his death on July the 15th, 1900, just over six months having elapsed since he was first thought to be ill, the diagnosis of chronic interstitial nephritis being confirmed by a partial *post-mortem* examination.

The history obtained was that as an infant the patient was very weakly; breast and bottle fed. During the first six months of life he suffered from a severe attack of eczema, which, however, under treatment entirely disappeared. From this time on till he was six years old the child, although never attacked with any definite illness, was constantly catching cold, was always white, and never seemed to put on flesh. His mother also noted that he used to pass large quantities of water; to this, however, she paid no attention. At the age of six he suffered from a mild attack of chorea, following on a fright. In the summer prior to his being brought for advice he got a very severe wetting, and had to remain in wet clothes for some hours; this, however, seemed to leave no bad effects. The next point of interest in his history is that his mother noticed that he had to get up several times during the night to pass water, which was light-coloured and frothy; also that he began to complain of headaches. His music teacher remarked that his eyesight was getting bad, he not being able to read his music nearly so well as he used to do. No particular attention was paid, however, to these points till his mother became alarmed at his pallor, thinness, and frequent headaches, and sought advice.

On examination the patient was found to be a very intelligent, bright boy. He had very pale, dark rings under his eyes, and a choreic-like movement of his lids. His skin was dry, inelastic, and distinctly dusky, especially about the top of his chest. His pulse was regular and hard. The apex-beat of the heart was just outside the nipple line. The first sound at the apex was impure, and there was distinct accentuation of the second sound at the base. The lungs, liver, and spleen were normal. The bowels were constipated. The urine was light-coloured, acid, of specific gravity 1006, and contained $\frac{1}{8}$th of albumen. There was no sugar. An examination of the eyes revealed œdema of the optic discs, if not actual optic neuritis. The patient was put to bed, placed upon a fish and milk diet, and a hot-air bath was ordered twice a week. Progress was very unsatisfactory; the headaches became more severe; acute abdominal pains set in, and the bowels were very constipated. The actual amount of urine passed varied from two to two and a half pints in the twenty-four hours, and its specific gravity ranged from 1006 to 1012. Albumen gradually increased in quantity until it reached $\frac{1}{6}$th, and hyaline and granular casts were found.

By the end of February the quantity of urine passed in the twenty-four hours increased to four pints, with a specific gravity of 1008, and albumen about a quarter. Epistaxis also set in, and under the skin on the small of his back there were small hæmorrhages. His pulse began to get irregular, and there developed a slight rise in the evening temperature. At times there were signs of improvement; but these were never lasting, and the patient gradually got worse.

By April the headaches were very severe, and abdominal pain increased. About this time also the patient was seized periodically with violent attacks of cramp in his legs, and these from now till near the end of his life played a large part in his symptoms. The urea output was estimated at this stage and found to be 2½ grains to the ounce, the passage of urine for the twenty-four hours being 70 ounces. An examination of the fundus oculi revealed albuminuric retinitis, hæmorrhages and white spots being visible. This examination was difficult, and I am sorry that the patient was not seen by an ophthalmic surgeon. Bronzing of the skin was also at this time a pronounced feature, being general over the body, with intensified patches. A dry, inelastic condition of the cuticle was also very noticeable. Hallucinations of various kinds were also present. He imagined that he could see all sorts of extraordinary things, *e. g.* a large number of Carter Paterson's carts following a funeral, etc.

By May his cardiac condition was much worse; a distinct double apical murmur had now developed, and the maximum apex-beat was one and a quarter inches outside the nipple line. Towards the end of this month he also had a slight convulsion, which lasted for three or four minutes. This was the only convulsion that occurred during his whole illness.

Early in June the urine began to decrease and the albumen to increase very rapidly. Œdema developed first under his lower eyelids and at the ankles, and gradually spread until it involved the whole of the body, producing a condition of general anasarca. The heart by this time practically fell "all to pieces;" epistaxis and hæmatemesis were at this time of frequent occurrence. The urine was passed at intervals of forty-nine, fifty-two, and forty-four hours, etc.

The boy gradually sank into a comatose condition, took but little nourishment, and died on July the 15th, a trifle over six months after he was first seen.

A full post-mortem examination was refused, but permission was granted to inspect the kidneys. The result of this examination was that both kidneys were found to be granular. The right kidney was very much smaller than the left, and it had a much dilated pelvis and upper part of its ureter. No obstruction at all could be discovered, and he thought the dilatation was explained by Dr. Goodhart's theory of atony. The kidneys were shown, as also a microscopical section from the left one, the condition being one of advanced interstitial nephritis. The renal tubules were in places completely obliterated by fibrous tissue. Some of the tubules were devoid of epithelial lining. In others the epithelium was present, and the blood-vessels showed great thickening of their walls.

The fact that such a condition in childhood is rare is undoubted. Dr. Guthrie, in the 'Lancet' of 1897, has recorded eight cases; another has been recorded in the 'Transactions of the Ophthalmological Society,' vol. xix, 1899, by Sutherland and Lawson. There are records of others in 'The Journal of Experimental Medicine,' vol. iv, 1899, p. 541, and in 'Archives of Pediatrics,' August, 1901, "A Case of Postero-sclerosis."

Dr. West, in his book on granular kidney, states that granular kidney may occur in the young, even in children; but though rare, it is still regarded as a much greater rarity than the case really is; and he has recorded an extreme instance at the age of eighteen years within his own experience.

The diagnosis in the above case presented no difficulty. With regard to his symptoms, abdominal pains and cramps played a considerable part. The headaches were such as are usually observed in such cases. One point of interest is the fact that only a single convulsion occurred, and that some six weeks before his death, and at a time when polyuria was well marked. Epistaxis was another troublesome symptom, and the hæmorrhages under the skin on the back were interesting. The condition of the skin, namely, the dryness, the inelasticity, and the brown discoloration, were well-marked features, and such alterations are said to be very characteristic of the complaint.

The toxæmic condition I take to be the cause of the pigmentation.

With regard to treatment I have nothing much to tell, save that it was symptomatic.

The point of greatest interest seems to me the question of causation—the ætiology of such a condition in children. Is the chronic condition the result of an acute interstitial nephritis, such a condition in children having been proved to be a fact by Holt ? If the result of an acute interstitial nephritis, then what is the cause of the latter ? If a chill were responsible, there is the history of a severe wetting taking place some twelve months previous to his death, but this was followed by no bad results apparently.

In the above case, however, if there were an acute interstitial nephritis, then I think it must be dated from infancy. In support of this there is the fact that the boy had never been strong, he constantly suffered from colds, he always kept pale and thin, and his mother noticed that he passed larger quantities of urine than was natural. Is it possible to connect the acute attack of eczema that he had, and which entirely disappeared under treatment, with the kidney trouble ? I am inclined to do so. The popular opinion that eczema may be "driven in" and set up graver troubles may have some foundation of truth.

Then such cases are said to have a syphilitic origin. Dr. Guthrie has discussed this question, and has arrived at the conclusion that syphilis is a very likely cause of chronic interstitial nephritis in young children.

In this case no syphilitic history could be obtained, and no syphilitic lesions were observed, unless the skin eruption be taken as possibly specific.

The idea that obstruction acted as a possible cause gains much credence in the above case from the fact that there was dilatation of the right pelvis of the kidney and of the upper part of the right ureter. As, however, no cause in the way of an obstruction to the flow of urine was discovered, it seems to me more reasonable to explain the dilatation on Dr. Goodhart's theory of atony.

Other causes have been advanced to explain chronic interstitial nephritis, but time does not permit me entering into them.

I am anxious to hear the views of some of the experts who are present, and who are much more qualified to express an opinion than I am.

(*Read January the* 17*th,* 1902.)

Discussion.

Mr. Sydney Stephenson said he noticed that retinitis was spoken of as being present; and he thought it quite possible that retinitis might be as common in interstitial nephritis in children as it was relatively in interstitial nephritis in adults. But since interstitial nephritis was very rare in children, it was likely that the non-frequency of retinal changes had been exaggerated. His own experience had furnished him with two such instances, the last of them a few days since. A boy aged 9 years was brought suffering from vertical headache with occasional attacks of vomiting and bad sight. His family physician thought those symptoms might be due to an uncorrected error of refraction—a not unreasonable supposition. When he (Mr. Stephenson) examined the fundus oculi, he detected the typical stellate figure of renal retinitis at each yellow spot. He found the urine was of low specific gravity, it contained a small amount of albumen, and included some granular and hyaline casts.

Dr. George Carpenter asked whether there was any history of syphilis in the family, and whether the arteries were hard during life. He thought from the notes which had been read it was a typical case of interstitial nephritis, and the microscopical section of the kidney shown confirmed that view. He took exception to the theory of "atony" which had been advanced as an explanation, and considered it a meaningless and therefore misleading term. He said Mr. Stephenson had spoken of ocular changes in such cases. He (Dr. Carpenter) had not yet had an opportunity of noticing any changes in the fundus oculi in cases of interstitial nephritis in children; but he remembered a case which came under his observation some years ago suffering from a typical large white kidney, clinically and *post mortem.* The age of the child, a female, was eight years, and it was the most marked example of renal retinitis he had observed. He had never seen it recorded in the text-books or elsewhere that in children renal retinitis was associated with the condition of large white kidney, but it was so in that case.

Dr. Sansom regarded the case as one of typical interstitial nephritis, but thought acute parenchymatous nephritis was grafted upon it. It was of very slow evolution, and the mode of death was not the epiphenomenon of arterio-sclerosis generally, but an engrafted parenchymatous nephritis, with œdema and the usual signs. But granted always that the ætiology was very difficult, they were nevertheless indebted to the author, because it was very valuable to get full records of such cases. He was inclined to endorse Dr. Carpenter's idea that anything like atony as an explanation was inconceivable. But one did know that certain very obscure forms of pyelitis were followed by slow-going interstitial nephritis. Such cases were recorded in Dr. Dickinson's book. They were exceedingly rare in young subjects.

Mr. Milligan (introduced), in reply, thanked the members for their criticisms. With regard to Dr. Sansom's suggestion about acute parenchymatous nephritis, he took the œdema in the case to be cardiac,—that it was due to failure of the heart's action. If the condition had been that of acute parenchymatous nephritis, blood would have occurred in the urine; but that was never found, although examined for repeatedly. Death he believed to be due to gradual exhaustion following on the toxæmic condition.

28. CASE OF THYROID DISLOCATION OF THE HIP-JOINT IN A CHILD.

By LEWIS W. MARSHALL, M.D.

The following case, the notes of which have been given to me by Mr. Dawson, our house surgeon, appear to me to be worth bringing before this Society, because the literature of this country scarcely refers to the possibility of such an occurrence in a child. This case is the only one I have seen of thyroid dislocation of the hip-joint, although its dorsal dislocation has not been uncommon in my experience.

Edmund Owen gives no instance in his surgical work on children's diseases.

Ashby and Wright say, "We have only rarely met with a traumatic dislocation (dorsal) of the hip in children" (ch. xxxix, p. 812).

Rose and Carless, in their latest work, when speaking about dislocations into the obturator foramen generally, express themselves as follows, viz.:—"The relative frequency of these dislocations (*i. e.* hip-joint) : Obturator 10 to 15 per cent. at all ages" (p. 446).

In 'Keating's Encyclopædia,' vol. ii, p. 1121, the statement is made that "cases of this kind (*i. e.* obturator dislocation) have repeatedly been observed in children." Twelve cases are cited. Of these Powdrell's at six months old is the youngest. Of the twelve, two cases are noted as doubtful.

My own case was typical, as the notes will show, and the X-rays confirmed the diagnosis.

Two points are noteworthy, the first being that a previous synovitis of the hip-joint had existed, and the second the simple way in which luxation was produced. The reduction was quite easily effected by manipulation, the pelvis being fixed by a colleague's hand, although the luxation was of more than four months' duration.

Mr. Dawson's notes.—Elizabeth P—, aged 9 years, was admitted to hospital on November the 13th, 1901. At that time she was unable to walk without assistance, and even then with difficulty. When standing her right leg was advanced, her body inclined forwards, the weight being supported on the front part of her foot with the heel slightly raised. The knee-joint was slightly flexed. Looked at from the front, the tensor vaginæ femoris and the adductor longus muscles were seen to be tense, and stood out very visibly. Viewed from behind, the buttock was found to be flattened, and a depression existed where the trochanter should have been. On measurement the limb was found to be lengthened by two inches. This lengthening appeared to be more, owing to the tilting of the pelvis towards the affected side. A thyroid dislocation of the hip was diagnosed.

The previous history showed that the child was taken ill with pain in the right hip-joint in March, 1901, and was treated by Dr. Polson, of Southwell, by rest in bed and a weight and pulley for "hip disease" during the succeeding three months. At the end of that time she was pronounced to be well, and ran about as usual. One day at the end of June whilst running on the pavement she slipped, and became lame from that time. Dr. Polson confirmed this history by letter.

A skiagram was taken by Mr. Simpson, of University College, shortly after her admission, and a few days afterwards Dr. Marshall reduced the dislocation by manipulation at the first attempt, a very obvious click being heard when the head of the femur returned to its socket. Mr. Morley Willis during the reduction fixed the pelvis with his hands, whilst the child was lying upon a mattress on the floor. The limb was then fixed by a long outside splint, with a large pad of wool on its inner side and a perinæal band. Four days after reduction the splint was removed and a plaster-of-Paris bandage applied, the knee-joint being included. The child soon returned home, being instructed to walk on crutches with a patten on the sound foot.

It should be stated that both limbs were symmetrical after reduction. (*Read January the* 17*th*, 1902.)

29. A CASE OF FRACTURE OF THE EXTERNAL CONDYLE OF THE HUMERUS IN A CHILD AGED NINE YEARS.

By J. JACKSON CLARKE, M.B.

The patient fell out of a cart in August, 1901, and was brought to me two weeks later. At this time the elbow-joint was all but fully extended and completely stiff. The original swelling had disappeared, but the joint had a clumsy appearance, which proved on examination to be due to outward dislocation of both bones of the forearm and backward dislocation of the ulna. The inner edge of the trochlear surface of the joint was prominent, the skin being stretched to whiteness over it. Radiograms confirmed the points previously observed, and, in addition, indicated that the external condyle, together with a narrow slip of bone which extended for some distance up the shaft of the humerus, had been fractured.

The patient was admitted into the North-West-London Hospital, where, under anæsthesia, the intra-articular adhesions were broken down, the joint moved to its full limits, and the displacements corrected. There was neither sound nor sensation of a true reduction, and the displacement returned when the force required to reduce it was relaxed. The limb was put up on an internal angular splint, a pad being placed above the internal condyle, and another below the elbow, to reduce the displacement to a minimum. Passive movement and massage were begun on the fourth day. At the end of the first week the wooden splint was replaced by a Croft's plaster-of-Paris splint which could be removed readily for daily exercising the limb. The pronation and supination movements were soon completely regained, and have so continued to the present time. After the plaster-of-Paris splint had been in use for three weeks it was left off for a time, with the result that the elbow-joint slowly became extended, and could not be flexed beyond a right angle, either by the patient's will or by any force that she would allow me to use. The plaster splint was then resumed, and I am now having made a light metal instrument that allows of free pronation and supination, but is fitted with a rectangular stop-joint which, while permitting flexion, does not allow extension beyond a right angle. By help of exercises, manipulations, and this instrument, I have no doubt that the function of the joint will improve considerably during the next year.

The case affords still one more example of how far from perfect the functions of the elbow-joint remain after fractures of the humerus in its neighbourhood. The question arises, how are such results to be avoided? The only answer is by better methods of immediate treatment. In adults I prefer to cut down, expose the seat of fracture, and wire the fragments, or do what is necessary. In children the effect of different positions of the joint should first be tried, and if a posture can be found in which the fragments remain in apposition, the limb should, I think, be put up in plaster of Paris in that position. The evidence of a radiogram may then be obtained. If by this means the joint is found to be in a bad position, a satisfactory setting can be obtained. I would suggest also that in children the open operation should be performed. (*Exhibited February the* 21*st*, 1902.)

Discussion.

Mr. Tubby said such cases of elbow injury were of great interest not only at the time, but afterwards, and sometimes of very painful interest to those who had attended them, as also to the patients themselves. He thought many of the old and bad results which had occurred were due to following the practice inculcated, which had been transferred from one surgical text-book to another. The books said, put the forearm midway between pronation and supination, and flex it at a right angle. But that advice was where the whole mischief in treatment originated. He did not know who attended the present case in the first instance, but, judging by the skiagram, if the arm had been forcibly extended at the time of the injury, then acutely flexed and fully extended, it would have been reduced, and the anterior part of the olecranon process would have made for itself a natural way into the front part of the fossa at the lower end of the humerus, and it would have been possible later on, by the weight of the arm, for extension to take place while preserving flexion. It would have been wise in that event to have tried originally by full extension first, succeeded by full flexion, to prevent the filling up of the two fossæ, and especially the anterior fossa at the lower end of the humerus. That was his practice in cases of injury of the elbow-joint, with one exception, viz. where there had been separation of the olecranon. The elucidation of the fact he had mentioned was largely due to Mr. Robert Jones, of Liverpool, who had taught him its value.

Mr. Jackson Clarke, in reply, thanked Mr. Tubby for his remarks, and said that if he got a primary case early he would act on his suggestion; but in a case which was not seen until two or three weeks after the accident, putting it into that position made no difference, owing to the callus which had already been thrown off.

30. A CASE OF CONGENITAL SPASTIC PARAPLEGIA.

By C. O. HAWTHORNE, M.D.

This patient, a boy aged 2½ years, exhibits in characteristic fashion the clinical phenomena of spastic paraplegia. There is extreme rigidity of the muscles of the lower limbs and heightened tendon phenomena. He is unable to walk without assistance, but when supported under the armpits he shows an extreme degree of "crossed-leg progression" as he attempts to move forwards.

With these evidences of an affection of the lateral columns of the spinal cord there are no mental or other symptoms of a cerebral lesion. There is no history of any kind of "fit;" the child is quite mentally acute for his years; he is of good temper and cleanly habits; and there are no athetoid or choreiform movements. The only possible qualification of the statement that there are no signs of disease of the brain is provided by the existence of some degree of convergent strabismus, and for this there is an alternative and more probable explanation. The optic discs are somewhat white but not definitely abnormal.

The question is whether the case is to be placed among the group of cerebral palsies in children, or whether it ought to be regarded as one of primary defect or degeneration in the crossed pyramidal tract. In addition to the absence, as noted above, of all evidences of a cerebral lesion, it has to be stated that there is no history of prolonged or difficult labour, or of asphyxia at the moment of birth—circumstances which are generally credited with a relationship to paralysis in infants by reason of their inducing changes in the cerebral cortex. So far from this being the case in the present instance, it appears that, in consequence of difficulty in the birth of a previous child, the pregnancy of which the patient is the issue was artificially terminated at the seventh month, and the child was then born easily and without recognised defect in regard to the function of respiration. In the absence of all other suggestion as to the cause of the paraplegia, the case may thus be quoted in support of the theory that premature birth may mean such a defect of the pyramidal tracts as to interfere with the adequate conduction of motor impulses from the cells of the cerebral cortex, with consequent imperfect power of voluntary movement, and uncontrolled activity of the lower motor neurons. In reference to the question of causation it must be mentioned that on two occasions during her pregnancy the mother had a somewhat serious fall. There is no evidence of syphilis; the family history is free from any record of nervous diseases, and the parents are not blood relations.

The circumstances of this case appear to demand the term congenital spastic paraplegia rather than that of cerebral paraplegia. However well founded is the application of the latter term to some cases of spastic paralysis in children, and however uncertain may be the theory of a primary degeneration of the pyramidal tracts, it still remains true that in the present instance there is no evidence of a cerebral lesion, and that the birth of the patient was not attended by the circumstances which are usually associated with such lesions. Congenital spastic paraplegia is a term which enters no doubtful area. That it implies imperfect conducting power, and doubtless also structural defect, in the crossed pyramidal tracts will be generally allowed; but whether such defect is a primary one, or is secondary to a lesion in the higher reaches of the central nervous system, is a matter of uncertainty. All that can be said is that to the latter view the facts of this case offer no support.

(*Exhibited February the* 21*st*, 1902.)

Discussion.

Mr. Carre-Smith asked whether there were any abnormal ophthalmoscopic signs in the case. The last case of the kind shown before the Society was by Dr. Garrod, and in that there was very well-marked retinitis pigmentosa. If good results with the ophthalmoscope in this case could not be obtained otherwise, he suggested an examination under chloroform. Eighteen months ago he showed before the West London Medico-Chirurgical Society two cases, sisters, one aged sixteen years and the other five and a half years. After careful examination the members came to the conclusion that they were due to syphilis, which was Dr. George Carpenter's opinion, from whose clinic he had obtained the children. The elder patient did not show any mental deficiency until six years of age, and the young patient when he saw her had no mental deficiency at all. He therefore concluded that in spastic paraplegia the mental weakness did not, as a rule, come on at first. There had been much discussion as to whether those cases were caused by prolonged labour, and in some cases they were. But he strongly held that the cases were of central origin, and did not originate in the spinal cord as in adults. He would be glad to hear if any other member thought the lesion was a spinal one.

Mr. Tubby said the best article on the subject which he knew of was one in 'Brain,' by Dr. Collier. Dr. Collier had picked out the main points of the affection in a most lucid manner. Regarding the mental development of those children, he had seen a large number of cases, and had tried to classify them mentally as far as possible. In none of the cases, he believed, could one say they were complete mentally. A certain number were good-humoured, placid, somewhat slow; others were very mischievous and petulant, and every now and then subject to passionate outbursts. Others were positively imbecile. From the surgical point of view the question was how far they should be touched by the surgeon. He was not accustomed to operate on those cases if they had athetosis or fits, or if they were markedly deficient mentally. On the other hand, he had seen considerable improvement mentally after operation, and had often noticed the mental condition improve *pari passu* with the physical. He would be glad to hear the differences pointed out between cerebral diplegia and double Little's disease.

Dr. Sutherland said that the presence of strabismus was a fact of great importance. He had seen a number of similar cases in which there was congenital strabismus, and where there was a certain amount of mental impairment. Looking at the conjunction of those symptoms in the present case, he thought that strongly confirmed the diagnosis of a cerebral lesion. The practical point was, that even a slight amount of congenital strabismus in a child was very suggestive of the diagnosis of the condition under consideration.

Dr. Hutchison said a perusal of Little's paper would show that he (of course) did not apply his own name to the condition, but he first described upwards of 200 cases of spastic paralysis, and they were of all kinds—some hemiplegic and others paraplegic. But he thought it would be admitted that since his time other authors who had used Little's name confined it to cases of paraplegia. He did not understand what Mr. Tubby meant by double Little's disease. As he understood diplegia, it was a condition implicating all four limbs, and differed from a double hemiplegia in the fact that the legs were more affected than the arms, and the rigidity tended to predominate over the paralysis. He thought those were the points laid down by Dr. Collier in the article in 'Brain' to which Mr. Tubby had referred, and which had been generally recognised in this country, and also in France and Germany, as marking off the two conditions. He was inclined to regard the case as one of Little's disease. With regard to ætiology it coincided with what Little found, that there was premature birth. The present child was born at the seventh month, and such a history was frequent in Little's disease. What was the causal relationship between that fact and the paralysis? Most people believed that the premature birth and the paralysis were both due to one cause—that the child was born prematurely because it or its mother was unhealthy or otherwise abnormal. But the labour in this case was artificially produced on account of the narrowness of the pelvis, and did not supervene on account of disease in the child.

Dr. James Taylor said his impression as to Little's disease had been the same as Dr. Hutchison's. Whatever Little may have described at first, his name had come to be associated, in this country and in America, with cases, like the present, in which there was paralysis affecting both legs, and usually affecting the lower limbs only. As to their origin there was very little evidence available. In the case of cerebral lesions the only evidence obtainable was in children practically born dead or who died almost immediately after birth. But that was unreliable, because many such children apparently lived. Two factors came into the causation. There was often a difficulty in the labour, or, at all events, an abnormality, and in the cases he had seen he was struck by the frequency with which the condition occurred in firstborn children, in whom presumably there was more difficulty in their entry into the world than in the later children. Where there was premature birth there was unusual celerity in the act. Doubtless congenital syphilis did come in, and he had seen two or three cases in which there was syphilitic choroiditis, whose symptoms were similar to those in Dr. Hawthorne's case. It was too early to speak of the mental condition of the case, and it was difficult to say whether there might not be at least mental weakness. He did not agree with Mr. Tubby that there was always some mental defect in such cases. In some of the patients, especially where the paralysis was confined to the leg, there was considerable intelligence, and one little girl under his care was preternaturally sharp. He had always thought that cases in which strabismus was present were much more liable to have mental defects; and he had associated that with the presumed position of the lesion, which was likely to cause strabismus. The centre subserving ocular movements was so far forward as to trench on the prefrontal area. Therefore, if there were a lesion in the area controlling ocular movements, there was much more likelihood of an affection involving the area associated with mental processes.

Dr. Hawthorne, in reply, said there were no structural changes in the fundus oculi. Though he had not been able to estimate the refraction, he had obtained a good view of the optic disc and other parts of the fundus, and nothing was present of the nature of retinitis pigmentosa. As far as the family history could be relied upon there was no suspicion of syphilis. In calling the case one of congenital spastic paraplegia he did not intend to raise the pathological question as to whether such cases were due to a cerebral or a spinal lesion. The reason he preferred to call it congenital spastic paraplegia was founded upon clinical propriety and security. The term could not be gainsaid in that case, whereas cerebral paraplegia involved a certain pathological speculation, which might or might not apply to his case. The term Little's disease had been for some years applied to cases of paralysis in the lower limbs.

31. A CASE OF HYPERTROPHY AFFECTING THE RIGHT LOWER LIMB.

By C. O. HAWTHORNE, M.D.

This boy is 10 years of age. He is the subject of considerable hypertrophy of the right foot and leg without any other recognised defect or deformity, excepting some degree of hypospadias. According to his mother's statement he has never been very robust, and he is a somewhat dull boy at school. But he has never had any illness excepting measles, and there is no history of injury. When from two to three years of age he is said to have been "bandy-legged." Shortly after this the limbs began to get straight again, and it was then observed that the right leg was longer than the left. The disparity has increased more particularly during the last few months, and the lad complains that his foot "throbs and burns," more especially after exertion. The patient is the fifth child in a family of thirteen, six of whom died in infancy; none of the others suffers from any malformation or peculiarity.

Examination shows very obvious enlargement of the foot and lower third of the leg, and, so far as can be judged, the hypertrophy affects the bones as well as the soft tissues. The right lower limb is 1¼th inches longer than the left, so that when the boy stands with his feet flat on the ground the pelvis is tilted. The measurement round the right instep is ¾th of an inch in excess of the left, round the right calf it is ⅞th of an inch in excess of the left, whilst round the thighs there is a difference only of ¼th of an inch. The skin of the right foot and the lower part of the right leg is unduly warm, the veins are prominent, and the pulsation of the arteries, both in front of and behind the ankle-joint, is very readily appreciated, the vessels apparently being dilated. On the dorsum of the foot is a hæmorrhagic patch measuring about a square inch, and a number of purpuric spots are present on the toes. Another patch, also hæmorrhagic, is seen on the front of the lower part of the leg; this presents a number of small vesicular prominences on the surface, and it is said that the other hæmorrhagic areas when they first appeared had similar characters. All these disturbances in the skin are of recent origin; they have not been present more than from three to six months.

The case accommodates itself to the general rule that in unilateral hypertrophy it is the right side which is enlarged, and the hypertrophy is mainly or solely manifested in the lower limb. There is very manifest disturbance of the local circulation, but this, so far as the history can be trusted, is of recent origin, the sense of pulsation in the foot and the hæmorrhagic patches in the skin having been observed only during the last few months, whilst the undue size of the limb was noticed several years ago.

Disturbances of the cutaneous circulation have been observed in several of the reported cases of unilateral hypertrophy. Such a condition existed in Finlayson's case, and also in some of the other cases of which he collected the records.[*] In at least one instance by A. N. M'Gregor an hypertrophy of one lower limb has been proved to be associated with a cerebral tumour.[†] There is nothing to suggest any such causation in the present instance, and it seems impossible to advance beyond the statement that the case is one of hypertrophy of the right lower limb associated with evidences of local vascular disturbance.

(*Exhibited February the 21st,* 1902.)

Discussion.

Dr. Sansom believed it was a genuine hypertrophy, not merely superficial. The right leg was larger than the left, and there seemed to be more articular development in it. All the tissues seemed to be hypertrophied on that side. He did not say otherwise than that there was also a superficial vaso-motor lesion, and no doubt the arteries were bigger and more in evidence on the right than on the left side.

Dr. Weber said he took a different view of the case from that of Dr. Hawthorne and from Dr. Sansom's. The patches on the skin were not hæmorrhagic, but nævoid or partly lymphangiomatous. The veins were much dilated, and it was a case which showed the connection between early varicose veins and capillary tumour-formation, a connection which Mr. Pearce Gould had recently referred to in his first Lettsomian Lecture before the Medical Society. As Dr. Hawthorne had mentioned, the arteries were also dilated, bringing the condition almost to one of cirsoid aneurysm of the extremity. Owing to the increased vascularity the limb had naturally grown more than its fellow. A child with nævus was shown at a previous meeting of the Society* by Mr. Roxburgh, and at the time he (Dr. Weber) privately drew attention to the great development of varicose veins in connection with the nævus. Cases of congenital varicose veins had been published, and they must have been of the same nature.

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[*] 'Glasgow Medical Journal,' November, 1884, p. 327.

[†] 'Transactions of the Glasgow Medico-Chirurgical Society,' vol. i, p. 130.

Dr. C. O. Hawthorne, in reply, said he could not agree with Dr. Weber. He thought some of the patches were certainly hæmorrhagic. The one on the dorsum of the foot he saw when the patient first came to him, and then it had the appearance of herpes. It was inflammatory, and he at first called it hæmorrhagic herpes. The patches had appeared only in the last few months, but the mother knew that the boy's limb was unusually long eight years ago. Thus the time incidence of the two conditions was a serious fact in opposition to Dr. Weber's view. Moreover the right limb was an inch and a quarter longer than the left. He did not think the lengthening was purely explained by the condition of the lower end of the tibia and bones of the foot.

32. CONGENITAL OCULO-MOTOR PARALYSIS.

BY SYDNEY STEPHENSON, C.M.

IN my own experience, congenital paralysis of the ocular muscles—with the possible exception of the levator palpebræ superioris—is far from common. I have, however, met with a few instances (certainly not more than half a dozen) of this form of paralysis affecting the external rectus. The following is the only case I have seen where palsy involved nearly all the muscles supplied by the third cranial nerve of one side.

Jessie H—, aged 6 years, came under my notice on December the 3rd, 1897. As her condition has not altered materially since that date, the following notes made at the time apply equally to her present state. She is the eldest of four children, of whom the patient herself is the only one that has ever been noticed to squint. Her father, who died at the age of twenty-nine years from "consumption and heart disease," had an inward cast of his right eye, said to have followed an attack of whooping-cough at three years of age. The squint was operated on, but without success, at Guy's Hospital. The patient was born at term, after an easy labour in which instruments were not used. Immediately after her birth the mother noticed that the right eye was closed, and although in a few months it became rather more open, still "it did not look right." When two years of age the child was taken to the Moorfields Hospital, where she attended for three months without deriving any benefit. She has always been delicate, and has suffered from measles, chicken-pox, and whooping-cough.

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* April the 19th, 1901. 'Reports of The Society for the Study of Disease in Children,' vol. i.

The child is poorly nourished, with slightly enlarged cervical glands. The knee-jerks are present; there is no ankle-clonus. Some degree of "habit spasm" is present. The urine is of specific gravity 1030, acid, with abundant urates, but free from albumen. She inclines her head towards the left shoulder when looking at objects. The right eye is directed upwards and outwards, with absent movements in every direction except outwards, and very slightly inwards. Rotation movements cannot be elicited. A large secondary deviation downwards and outwards appears in the other eye when the affected eye is made to "fix" the finger. There is no ptosis, and the eye can be closed readily and completely. Double vision cannot be elicited. The right pupil, which is pear-shaped, measures 5 mm. transversely. It has no direct action to light, and its "indirect" or "consensual" reflex appears also to be lost. It becomes smaller after the local application of physostigmine and larger after homatropine. R. V. $\frac{6}{60}$, $\frac{+\ 1{\cdot}5\text{ D. sph.}}{+\ 2{\cdot}0\text{ D. cyl. }90^\circ}$ $\frac{6}{18}$. The child can read with the affected eye only the large letters of Jaeger's hand-type (No. 14), having a height of 5 mm. With a + 4 D.

spherical lens added to the ametropic correction, she reads the smallest type at a distance of 25 cm.; with a + 5 D. spherical lens the same type is read at 20 cm., and so on. There is, therefore, complete paralysis of accommodation. The fundus oculi is normal, although the retinal vessels, especially the veins, are tortuous. The pupil of the left (unaffected) eye measures 3 mm., and responds well and promptly to light, darkness, and convergence. It also acts consensually, as when a beam of light is thrown into the fellow-eye. L. V. $\frac{6}{9}$ and No. 1 J., $\frac{+\ 3{\cdot}5\ \text{D. sph.}}{+\ 0{\cdot}5\ \text{D. cyl. } 90^\circ}\ \frac{6}{9}$. The fundus oculi is normal, save for tortuous retinal vessels.

No palsy of any of the other cranial nerves exists.

Remarks.—In the foregoing case it is evident that the third cranial nerve on the right side is totally paralysed, except as regards the branch which supplies the levator palpebræ superioris muscle, which seems to have been involved at birth, but to have recovered its functions a few months afterwards. The branch which supplies the internal rectus muscle also has partly escaped. It is a question, too, whether the superior oblique muscle supplied by the fourth cranial nerve retains any action.

There are two views with respect to the ætiology of these congenital defects in the ocular movements: either that they are due to an absence, an ill-development, or an abnormal attachment of the muscle affected; or that they are the result of an absence or functional inactivity of the nuclei of origin of the nerves supplying the affected muscle or muscles. In favour of the first or muscular theory there is a certain amount of direct evidence. For example, Dr. Heuck * obtained a post-mortem examination of the body of a young man aged eighteen years, who during life had exhibited ptosis with absence of the upward and downward movement of the eyeballs, and defective action of the internal and external recti muscles. At the autopsy the levator palpebræ superioris muscle was badly developed on the right side and absent from the left, thus accounting for the ptosis observed during life. The imperfect movements of elevation and depression were apparently due to an abnormal insertion of the superior and inferior recti muscles. Indeed, in this remarkable case every extrinsic muscle of the eyeball with the exception of the internal rectus was inserted into an abnormal part of the sclerotic coat of the eyeball. Somewhat analogous cases have been reported by Steinheim, Baumgarten, Olbers, and Diffenbach.* That the muscular theory cannot apply universally is clearly shown by a case of my own, where the external rectus was found to be present in a patient with congenital convergent squint. The facts are briefly as follows.

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* 'Klin. Monatsbl. f. Augenheilk.,' 1879.

Florence F—, aged 16½ years, came to me with a large congenital convergent squint of her right eye measuring forty degrees on the perimeter. Abduction of the eyeballs was imperfect, more especially on the right side. Nystagmus was not present; diplopia could not be elicited. Chloroform was administered, and the internal rectus muscle of the squinting eye divided. The external rectus muscle was then exposed, and found to have a normal appearance and natural attachment to the sclera. That muscle was shortened, and sutured to a new attachment on the sclera nearer the cornea. The remote result of the operation was noted as excellent.

The muscular theory of causation has the merit of simplicity, and is doubtless capable of explaining satisfactorily many of the cases where a single ocular muscle is congenitally paralysed. But its shortcomings are manifest when an attempt is made to apply it to a case such as that shown this afternoon. Indeed, it would be a strange coincidence if a paralysis affecting practically every branch of the third nerve could be produced by malformation or absence of the several muscles, external and internal, supplied by that nerve. In such a case the alternative or nervous theory must, I think, be adopted.

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* 'Trans. Ophthal. Soc.,' vol. viii (1888), p. 271.

(*Exhibited February the* 21*st*, 1902.)

Discussion.

The Chairman (**Dr. James Taylor**) said such cases were very rare. He did not know whether any definite lesion had been found in the nerve nucleus.

Mr. Carre-Smith asked whether there was any suggestion that it had been caused by specific disease, because the only case he had seen like it was in an adult, and it was not congenital, but was due to a gumma pressing on the third nerve. Under iodide of potassium the paralysis cleared up in a short time.

Mr. Stephenson, in reply, said the patient was the oldest of four children (living); there was no history of miscarriages or still-births, and there was no evidence about her of congenital syphilis.

33. PERFORATION OF A TUBERCULOUS ULCER OF THE SMALL INTESTINE.

By E. W. GOBLE.

(Introduced.)

E. B—, a boy aged 6 years, was admitted to the North-Eastern Children's Hospital on October the 15th, 1901, under Dr. W. A. Wills, suffering from diarrhœa, cough, wasting, and a distended belly, of two and a half months' duration. The diagnosis made was general tuberculosis. His condition remained stationary till October the 25th, when he was suddenly seized with symptoms of acute general peritonitis. At the autopsy tuberculosis was found to be general. A perforated ulcer three feet above the ileo-cæcal valve was discovered, and fæcal matter was found in the peritoneal cavity.

(*Exhibited February the* 21*st*, 1902.)

Discussion.

Dr. Gwynne (Sheffield) said that such tuberculous nodules on the intestine sometimes existed without producing symptoms.

In operating for the radical cure of hernia he had found the peritoneum covering the gut full of those nodules, and yet the child was apparently healthy, and made a good recovery.

34. A CASE OF HYPERTROPHIC CIRRHOSIS OF THE LIVER IN A GIRL AGED TEN YEARS.

By W. C. CHAFFEY, M.D.

THE patient was admitted under my care at the Children's Hospital, Brighton, on November the 18th, 1901. The mother stated that the child was illegitimate. It had bronchitis at four months of age, and had contracted measles and whooping-cough, but not scarlatina or typhoid fever. She was given alcohol occasionally when ailing, but not habitually or in excess. The mother also stated that she had had no miscarriages, and that none were born dead. She herself enjoyed fairly good health, but the child's father was not strong (? phthisical). The patient was under my care two years previously, when her condition was much the same as at the present time. Since then she had on one occasion vomited about a tablespoonful of blood.

On admission she was found to be poorly nourished. Her complexion was sallow, with a slight icteric tint of the conjunctivæ, and patches of enlarged venules on the cheeks. There was no dropsy anywhere. Her temperature and pulse were normal. The abdomen was rather prominent at the epigastrium, where there were some enlarged superficial veins. The liver could be felt reaching nearly to the navel, and it was very firm, and knobby on the surface. The spleen could also be felt, and was much enlarged. An examination of the chest revealed nothing abnormal.

On November the 28th the patient was placed on Potassium Iodide grains iij, Liquor Arsenicalis minims iij, thrice daily. Mr. Fairhall, the house-surgeon, made an examination of the blood, and reported 78 per cent. of hæmoglobin, red cells, 5,170,000 per c.mm., and no increase of leucocytes. The motions were normal in colour, and the icteric tint of the conjunctivæ was not marked.

On December the 3rd an examination of the abdomen revealed a considerable diminution in size of both the liver and spleen. The patient became very drowsy. The urine contained an excess of bile pigments.

December the 8th.—The patient has remained very drowsy, but does not complain of any pain. There is some tonsillitis, accompanied by a slight rise of temperature. The urine is of specific gravity 1026, acid in reaction, smoky in appearance (blood), and free from casts.

December the 11th.—Yesterday the patient developed vesical incontinence; she swallows well, but is very drowsy. The right pupil is dilated and fixed. The knee-jerks cannot be obtained, and the left hallux gives Babinsky's sign. There is slight left ankle-clonus.

Post-mortem examination.—Both the liver and spleen were much enlarged, the former being everywhere traversed by bands of fibrous tissue, and its substance everywhere more dense than was normal. Both kidneys were greatly enlarged and congested, but apparently not otherwise affected. The base of the brain showed patches of recent lymph, especially in the interpeduncular space. There was no tubercle visible anywhere. Microscopical sections of the liver revealed perilobular cirrhosis, the liver cells showing but little departure from the normal.

Remarks.—The cause of the cirrhosis seems difficult to explain. In favour of congenital syphilis there is the fact that the child was illegitimate; but, on the other hand, there is nothing pointing to this disorder in other members of the family. The mother is a quiet, well-conducted woman, and does not appear to be one who would dose her children largely with alcohol. The cerebral symptoms were a peculiar feature. It is difficult to explain the effusion of plastic lymph at the base of the brain. The microscopical section of the liver tends to suggest alcohol rather than syphilis as the cause.

(*Read February the* 21*st,* 1902.)

Discussion.

Dr. Lister said the case seemed to him to raise the question of an origin of cirrhosis in children other than alcohol and syphilis. In the 1900 meeting of the British Medical Association there was a discussion on the pathology of cirrhosis, introduced by Dr. Voelcker. Professor Flexner was present, and described acute interstitial hepatitis produced in guinea-pigs by the injection of toxines into the liver. At the same meeting he (Dr. Lister) had shown specimens from a boy who died after ten days' illness from acute ulcerative enterocolitis with hæmorrhage. His liver showed a remarkable interlobular infiltration of small cells with much destruction of the liver cells by hyaline degeneration and vacuolation. Flexner had produced similar conditions by injection of special strains of the colon bacillus, and he (Dr. Lister) thought that in some of the cases of acute enteritis in children an explanation might be found of some of the obscure cases of cirrhosis of the liver.

The Chairman (Dr. James Taylor) said he saw a case of extreme cirrhosis of the liver in a girl of fifteen, who was at the Queen Square Hospital on account of nervous symptoms and choreiform movements. The discovery in the liver post mortem was all that could be found, and it was a great surprise. There was neither history of alcoholic indulgence nor of fondness for spices. He thought there was a class of cases of cirrhosis of the liver of which little was known, either as to origin or symptoms.

Dr. Weber asked whether the cirrhosis of the liver was discovered by chance, and whether the patient died definitely of meningitis. The size of the spleen also had a bearing on the subject. Probably the child had an independent brain disease, and, but for that, would have lived much longer.

Dr. Sansom said he hoped a record would be kept of Dr. James Taylor's remarks about cerebral symptoms in genuine cases of cirrhosis. The discussion went far to establish the great importance of the Society in getting together cases in children which would exemplify disease in general. He remembered a case at the North-Eastern Hospital for Children of cirrhosis of the liver in a child, about which a so-called diligent inquiry elicited nothing, but a further inquiry revealed the fact that the child had been supplied daily with considerable quantities of gin in teaspoonfuls. Cirrhosis of the liver afforded a very good example of sclerosis; but sclerosis might occur at many points, and the symptoms mentioned by Dr. Taylor might have been due to some cerebral sclerosis.

35. A CASE OF PAROXYSMAL HÆMOGLOBINURIA IN A BOY AGED THREE YEARS.

By GEORGE CARPENTER, M.D.

Benjamin L—, aged 3 years, was brought to the out-patient department at the Evelina Hospital on January the 15th, 1902. His mother found his urine to be port-wine coloured the day before; he cried when he began to pass water, and he complained of pain in the hypogastric region. Subsequently on several occasions his urine was found to be dark red coloured, usually in the afternoon. Its passage was attended by preliminary pain, sometimes a quarter of an hour beforehand, and subsequently similar urine was passed painlessly. He had been noticed to "shiver" and "go blue" before voiding such urine.

A spectroscopic examination of the diluted hæmoglobinuric urine revealed two absorption bands between the solar lines D and E in the yellow and green. An undiluted specimen showed an absorption band in the red, with obliteration of the rest of the spectrum. Oxalates were not present. The boy had not been out of England; he lived in New Cross; there was no history of syphilis, and nothing to suggest that disorder on examination. He was a comparatively healthy child with warm extremities. He had suffered somewhat from rickets, there were palpable lymphatic glands in the neck and groin, and the free edge of the liver was 1½ inches below the costal margin in the nipple line.

He was subsequently admitted into the wards for further observation, and during his stay there the urine was healthy. On February the 15th his left forearm was tightly bound with a rubber catheter, so as to stop the circulation. His left hand was then immersed in ice-cold water for five minutes. A blood examination of the right hand showed red corpuscles 4,800,000 per c.mm. Of the left hand, after five minutes' immersion red corpuscles numbering 3,783,000 per c.mm. were counted. Thus after five minutes' topical application of cold there had been a destructive action to the red corpuscles of over 20 per cent. Hæmoglobin was not detected in the urine.

On February the 20th the right leg up to the middle of the thigh was immersed in ice-cold water for five minutes. A blood-count was not made on this occasion. The child passed no dark urine after the experiment, nor was there a rise of temperature.

(*Exhibited February the* 21*st*, 1902.)

Discussion.

Dr. C. O. Hawthorne asked whether there was any evidence of syphilis in the case. Paroxysmal hæmoglobinuria had been observed in a considerable number of cases of acquired syphilis and in a few cases of the inherited disease. As far as he knew, its association was mainly with enlargement of the spleen, but evidently that was not present in Dr. Carpenter's case. One such case was recorded by Mr. Hutchinson in his article on 'Congenital Syphilis" in the 'Twentieth Century Practice of Medicine.' A case was also recorded some years ago by Mr. Lawford Knaggs, in which the child gave no evidence of congenital syphilis other than paroxysmal hæmoglobinuria. Some years ago he himself had a case of enlargement of the spleen and paroxysmal albuminuria. The boy on one or two occasions became chilled, had rigors, and passed urine containing albumen.

The Chairman (**Dr. James Taylor**) said that at the North-Eastern Children's Hospital he had a case of typical paroxysmal hæmoglobinuria without any sign of congenital syphilis. But two months ago the patient came to the hospital on account of a sore throat, and then he found that her uvula was almost completely eroded and the soft palate much invaded by what was no doubt syphilitic ulceration. In that case, at Dr. Sutherland's suggestion, he looked for oxalates in the urine, and found them. The oxalates remained after the subsidence of the hæmoglobinuria.

Dr. Sutherland said he could not settle what was the significance of the oxalates in such cases, but it was his experience that they were present. They were more excessive in the boy mentioned by the Chairman than in any other case he had seen. The oxalates would persist for weeks after an attack.

Dr. Weber said the practical point in the paper was the importance of shielding the child from the exciting causes of the attack. If possible, such a child should be reared in a warm climate, in which there was little risk of a sudden chill. He thought oxalate crystals could be found in nearly every case in which blood-pigment, either with or without blood-cells, came away in the urine. The only case he had seen of paroxysmal hæmoglobinuria in a child was in a subject of Raynaud's disease, and with signs of congenital syphilis.

A Member said a man was brought to hospital suffering from violent abdominal pain, and with cold lower limbs. He passed urine of a port-wine colour; it contained no blood-discs, but hæmoglobin was revealed by the spectroscope. He had great anæmia, and died three months later, when it was discovered that he had an aneurysm of the abdominal aorta, from which a plug had become detached and had blocked up both iliac arteries. In that case the lower limbs being practically cut off the blood was crowded into a smaller space, and the blood-discs were probably broken down to make room.

Dr. George Carpenter, in reply, said in the case of hæmoglobinuria which he showed at the last meeting no evidence of syphilis in the child could be obtained, but he had previously treated an infant sister for congenital syphilis, and there was a history of that disease in the family. It was very difficult, after one year of age, to detect the syphilitic taint; indeed, obvious syphilitic lesions were not often seen after the first six months of life. Until the eruption of the test teeth, or the development of other characteristic lesions, and the stamping thereby of the disorder, the syphilitic taint was usually accidentally discovered as it were, and not by reason of the assistance offered to diagnosis by the presence of obvious stigmata. In the present case there was nothing to suggest syphilis, either by inquiry directed to the family history or from facts gleaned by a physical examination of the patient, but notwithstanding that the taint might be there, though not in evidence. No doubt cold killed the red blood-corpuscles wholesale.

36. A CASE OF ICHTHYOSIS IN AN INFANT AGED FIVE MONTHS.

By GEORGE CARPENTER, M.D.

Charles B—, aged 5 months, was born with a healthy skin, with the exception of one eye and a small piece of the nose which would not wash clean. Within a few days of birth the whole of his body "appeared like that of a very aged person." It was "wrinkled and began to peel."

He is the first and only child. There have been no miscarriages or still-births. A month from his birth he developed "snuffles."

The infant's skin is now a mixture of white and a light oak-stain colour, the latter a discoloration from dirt, and cod-liver oil which has been freely applied. The oak-stain coloured areas are covered by an adherent parchment-like epidermis. On raising the edges of these patches, which tend to curl up, the underlying parts appear raw-looking and weep. Over the white skin-like areas the cuticle is harsh to the feel, and slightly roughened; it is indistinguishable from the condition of skin known as xerodermia. The dirty, adherent, thickened epidermis-bearing areas are largely in excess of the white and more healthy-looking parts, and in many places show cracks and fissures which by their intersections map out lozenge-shaped areas. The scalp, which is wrinkled and has an ill-nourished appearance, is free from hair, and in parts has patches of thickened, dried epidermis attached to it. The eyebrows are lost in dirty, thickened epidermis.

There is very extensive craniotabes over the whole of the parietal bones. The eyes are red and weak-looking, and have been discharging. The eyelashes are normal. The face and neck are invaded with thickened epidermis-bearing areas in patches. Of the rest of the body the trunk is the worst, and the extensor surfaces of the trunk and limbs are more involved than the flexor. The soles and the palms have not escaped. The finger-nails are brittle, but the nails of the toes are fairly well formed.

There is a muco-purulent discharge from the nostrils, and "sometimes the ears discharge matter." The organs are normal. The ends of the long bones are not enlarged. There is a left inguinal hernia. He perspires "very rarely;" the mother has noticed it "where the skin has come off." The urine was drawn off by a catheter; there was a very faint trace of albumen in it.

(*Exhibited February the* 21*st*, 1902.)

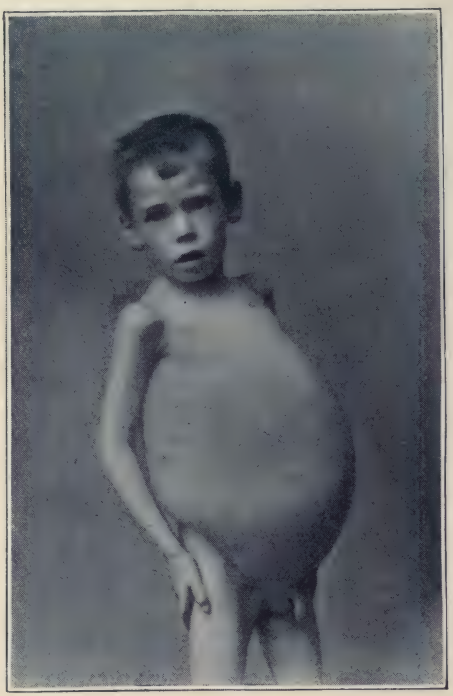
37. CASE OF CONGENITAL DILATATION OF THE SIGMOID FLEXURE OF THE COLON.

By C. N. GWYNNE, M.D.

On September the 11th, 1900, W. H—, a lad aged 6½ years, was admitted into the Sheffield Children's Hospital. The following history was obtained from his mother:—She had nursed him up to the age of twelve months. From birth he was remarkably constipated. She had frequently to administer enemata, with but slight result, and, in fact, he never had a natural evacuation. The constipation increased until one evacuation in three weeks became the rule. He had occasional vomiting, which was relieved by the passage of hard scybala by the bowel, followed by some diarrhœa. His abdomen, which had always been large, gradually increased in size, while elsewhere he grew emaciated.

On admission the boy seemed in a sort of stupor, lazily answering questions, and lying like a log in the bed. He even took no notice of rather a lengthy digital examination *per anum.* The abdomen was enormously distended, measuring thirty-one inches just above the umbilicus (*vide* Fig. 16). Big coils of bowel could be plainly discerned through the abdominal wall. The rectal examination gave no information. For two days repeated injections were given, accompanied by massage of the abdomen, with no result. As his condition seemed dangerous from extreme exhaustion, I determined to perform colotomy to relieve any mechanical obstruction that might exist, or, at any rate, to provide an escape for the pent-up fæces. With a view to a better examination of the abdominal cavity, I made my incision in the mesial line from the umbilicus towards the pubes. Enormous coils of intestine at once presented at the wound, and appearances denoting general chronic peritonitis were present. The whole length of the colon from the cæcum to the rectum was enormously distended, and from its size and the adhesions formed about it could hardly be moved. The greatest dilatation was at the sigmoid flexure, where it must have been not less than four inches in diameter. After ascertaining so much I quickly closed the wound, and decided to make an artificial anus in the left iliac region, which would be a more convenient position if it had to be kept open for any considerable period. The bowel in this region I found firmly bound down by adhesions, and its walls were greatly thickened, apparently a true hypertrophy. After the operation a continuous flow of fæces of the consistence of a poultice took place for about ten days, and the size of the abdomen became greatly reduced.

Fig. 16.



After two or three months' duration a gradual return to a normal evacuation began, and less came through the artificial anus. His intelligence, too, increased, and that impassive, listless state which was so evident when he was admitted into the hospital gave place to a more alert state of mind. Unfortunately in the following spring he contracted scarlet fever in the hospital, and had to be removed to Lodge Moor Fever Hospital. Last October I admitted him again into the hospital, and the abdomen was nearly, if not quite, normal in appearance; the motions, though still irregular and at times constipated, came *per vias naturales.* The artificial anus was practically closed. The general health of the boy was excellent, and his body was well nourished.

About a month after the operation I made several examinations of the bowel with the aid of large bougies, and was enabled to map out accurately the course of the large intestine from the sigmoid flexure to the anus. I ascertained that the sigmoid flexure close to the pubes took a sudden upward direction, almost at a right angle to itself, as far as the level of the umbilicus; here it doubled backwards upon itself, and pursued an almost straight course to the anus. Up to what may be called the pubic turn, the bowel was enormously distended, this abrupt turn opposing a strong barrier to the progress of the bowel contents. In the portion bounded below by the pubic sharp turn and by the acute turn upon itself in the neighbourhood of the umbilicus the gut was not so dilated; but here a hard mass of fæces had collected, which had become imprisoned, and it was only with the greatest difficulty that I at last succeeded in breaking up this mass and getting it evacuated, partly *per anum,* and partly backwards through the opening in the groin.

Dr. Herringham* has contributed a most interesting article in connection with a case of idiopathic dilatation of the sigmoid flexure that occurred in an old man aged 70 years. At the *post-mortem* examination of this case the sigmoid flexure was found enormously distended, and resembled a dilated stomach turned upside down. Elsewhere there was nothing abnormal found. In three other cases occurring in patients over fifty years of age similar *post-mortem* appearances were found, the sigmoid flexure alone being affected. All these cases were the subjects of obstinate constipation. The other cases recorded occurred in young subjects, and seem to belong to quite a different category. In them the troubles began or were noticed in earliest infancy, and the dilatation affected different parts of the large intestine, the sigmoid flexure, however, being the most usual site. In December, 1900, Dr. W. L. Stowell, at a meeting of the New York Academy of Medicine, showed a boy, aged 9 years, with great dilatation of the colon which could be seen through the abdominal walls. The abdomen at the umbilicus measured 28½ inches.

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* 'St. Bartholomew's Hospital Reports,' vol. xxxi, 1895.

In 'Pediatrics' for May of last year Dr. Theodore Fisher, Pathologist to the Bristol Royal Infirmary, contributed the history of two cases. One was a male child aged 9 months. He was very emaciated, and the abdomen measured at the umbilicus 23½ inches. Large distended coils of colon could be observed, and "huge vermicular movements" at the left side of the abdomen. At the necropsy the large intestine was found greatly dilated, chiefly in the transverse colon and sigmoid flexure. The cæcum and ascending colon were unaffected. The second case was that of a female child aged 13 months. At the *post-mortem* the transverse and descending colon were found enormously dilated, the diameter of the transverse colon being 5 inches.

There are so very few cases presenting this condition on record that you will pardon me if I draw your attention to two other cases, as they are especially instructive. In one the patient was a boy aged 11 years under the care of Dr. Walker. The condition was noticed soon after birth. The distension of the abdomen was enormous, being 27 inches at the level of the umbilicus. The transverse colon at its largest part was 16 inches in diameter. The dilatation ended in the middle of the sigmoid flexure.

Dr. Griffiths, Pathologist at Addenbrooke's Hospital, Cambridge, who made a *post-mortem* examination of this case, thinks the series of events began in an acute colitis, subsequently becoming chronic ; great distension followed from the chemical changes in the fæces, much gas being produced. The distension that followed would naturally render peristalsis difficult. This difficulty would explain the great increase in the size of the muscular fibres, and also the formation of new fibres; in fact, a true hypertrophy.

In December, 1895, Dr. Church admitted a case into St. Bartholomew's Hospital. The patient was a female child aged 4 months. The distension extended from the middle of the ascending to the upper part of the descending colon. It began and ended funnel-wise. There was no kinking or external compression.

A general review of the recorded cases, which time and space have not permitted me to trace in detail, has impressed upon my mind the following conclusions :

1. That while the sigmoid flexure seems to be the seat of selection, any portion of the large gut may be involved, the rectum being an exception.

2. The affection is by no means necessarily congenital, though some cases point to an origin as early.

3. That it is not due to kinking, pressure by bands, or other external mechanical cause.

4. That constipation is present in every case, but that it is a concomitant or resulting condition, and in no respect the cause. If such an every-day condition as constipation were the cause, we should find frequent examples of the disease, while in fact it is one of the rarest.

5. That colitis with inflammation extending to the muscular coat, causing paralysis and subsequent distension, and still later hypertrophy of the muscular coat, explains a certain number of cases.

6. That some cases seem, from their history, to point to some congenital peculiarity up to the present not elucidated.

7. That the prognosis is bad.

(*Read February the* 21*st*, 1902.)

38. THE LOCAL TREATMENT OF ADENOIDS.

By J. PORTER PARKINSON, M.D.

I HAVE ventured to bring before the Society to-day a plan of treatment which I have employed for the last three or four years at the North-Eastern Hospital for Children and elsewhere for a certain class of case usually called adenoids.

The children affected suffer from periodic "colds," generally spoken of by the mother as "bronchitis;" they have attacks of violent coughing, often worse at night or in the early morning, and perhaps followed by vomiting; they often have discharges from the nostrils or ears, with slight deafness, occasional headache, and suffer, if of a neurotic temperament, from night terrors.

On examining the chest nothing abnormal may be found, except a slight snoring inspiration and an occasional rhonchus.

The child may have the facial aspect, snoring respiration, and nasal speech which denote adenoids, and on examining the naso-pharynx with the mirror, or preferably with the finger, the swelling known as adenoids may be felt; in some cases this swelling is hard and fibrous feeling, but in a large proportion it is soft and gelatinous. In the former case there is obviously but one hope for a permanent cure, that is complete removal of the growth, especially if nasal breathing be much interfered with, or complications such as deafness be present.

In these cases, however, where a soft and gelatinous mass is to be felt the line of treatment is not so obvious; if left alone, the child in a week or two with ordinary care may improve, but the obstruction to nasal respiration will to some extent remain. In these cases, which Sir F. Semon in his recent lectures on "Local Treatment of the Upper Air-passages" alludes to as "periodic and transitory," but little has been suggested to hasten the disappearance of the catarrhal symptoms and to relieve the underlying condition. In them, as Sir F. Semon has pointed out, there may be only a very moderate, or hardly any, organised hypertrophy of the lymphoid tissue, but on the slighest provocation so much congestion and engorgement occurs that for the time all the symptoms of genuine adenoids are closely simulated. In such cases as the above I have been in the habit of applying locally to the naso-pharynx an astringent solution, with the object of producing shrinking of the engorged tissue, so as to allow of nasal respiration being almost at once commenced, and to prevent, if possible, any permanent thickening being left behind owing to the long continuance of a chronic congestion.

I am aware that there is nothing new in this. Mr. Urban Pritchard has suggested the injection of a solution of tannic acid or a spray containing the same substance by the anterior nares; but it has always appeared to me that the direct application to the part chiefly affected would be more likely to ensure success, and could have no bad effect on the mucous membrane of the nostrils. Consequently, after satisfying myself by digital examination that the obstruction is of the soft gelatinous variety, I apply a solution consisting of equal parts of Liquor Ferri Perchloridi and glycerine by means of a brush attached to a curved handle, the most convenient being the laryngeal brush made by Meyer and Meltzer, which can be bent to such a curve that it can easily be introduced behind the soft palate and the naso-pharynx thoroughly painted. With a little practice this can be done rapidly, and with such a minimum of discomfort that the majority of children do not fear its application. This may be done twice weekly at first, and continued once a week till all the symptoms have abated. I supplement this with respiratory exercises, such as those suggested by Mr. Arbuthnot Lane, and the administration of a mixture containing cod-liver oil and iron, and I believe the symptoms disappear much more rapidly than in cases not locally treated, and the after-history shows that in a large majority of the children the symptoms have but little tendency to recur, and nasal respiration is permanently established.

In the slighter cases improvement begins after the first application, but many have to be painted oftener before the symptoms abate; but when improvement begins it is surprising how permanent it is, for one would naturally expect that the astringent effect would be only temporary. This may perhaps be attributed to the prevention of that permanent thickening which results from frequent or long-continued congestion. It is generally admitted that a large proportion of adenoids atrophy spontaneously about the period of puberty, and therefore the nearer the child is to that age the less permanent is an obstruction likely to be; but it is by no means these cases alone which are benefited by the astringent treatment, for it seems equally efficacious in children under the age of ten years.

In those cases where permanent obstruction is present, and the naso-pharynx is blocked by a tough resistent growth, this treatment is naturally of no avail, and removal of the growth is imperative; but in suitable cases the simple method described will be found to give satisfactory results. Again, in cases which have undergone operative treatment, sometimes more than once, and in which the symptoms have recurred, I have found great benefit ensue. (*Read February the* 21*st,* 1902.)

Discussion.

Dr. Sutherland said he had no experience of the treatment recommended, but with regard to the nature of the treatment in connection with tonsillitis the method of painting had been employed for many years, and he thought it had been given up for some time. The substance of an enlarged tonsil was much the same as that of adenoids, and one would be sceptical of a treatment for adenoids which did not answer for enlarged tonsils. In connection with soft tonsils he (Dr. Sutherland) found that painting was practically of no use. Did Dr. Parkinson find soft tonsils subsided under the treatment?

Dr. Parkinson, in reply, said Dr. Sutherland appeared to have mistaken the kind of cases referred to, which were those in which there was little or no organised hypertrophy, but purely a passing condition of the naso-pharynx, especially a catarrhal condition, which was probably induced by a slight increase of lymphoid tissue, successive increases producing marked symptoms. By preventing a child from suffering from periodic colds much might be done to keep away a condition of real adenoids. He had used the term "adenoids" because no other would include the condition he meant. He agreed that "adenoids" was a much misused term. He thought the reference to enlarged tonsils in that connection was not quite relevant.

39. A CASE OF CHRONIC HYDROCEPHALUS.

By GEORGE CARPENTER, M.D.

Arthur L—, aged 8½ years, had a small head at birth. He was an "eight months baby," and weighed 9 lbs. at his birth. He was healthy until fourteen weeks old, when he contracted diphtheria, scarlet fever, and pneumonia. During his illness he had a "fit" which lasted two days, and "he lay as if dead." After this there were vomiting, wasting, head retraction, and some opisthotonos. His "sight was good." At this time his head began to increase in size. At two years of age he had scarlet fever and diphtheria and pneumonia, and paralysis of the soft palate. After this his head was much larger, and has since continued to increase in size. He began to talk at sixteen months old, and to walk a year ago. Five children of a family of six are alive and well. All had "snuffles" when infants.

The child's head is obviously enlarged, and measures 24½ inches at the greatest circumference. The bony prominences are exaggerated, the parietal eminences and the occipital protuberance being especially well marked. There is no thinning of the cranial bones. The fontanelles are closed. The orbital plates are not thrust down, and the optic axes are not altered.

He can say his A B C with an occasional hiatus, and he can count up to sixteen. He has rarely attended school. The fundus oculi is natural, and his sight is good.

His hands are large and flabby, and their movements clumsy. There is no rigidity of the arms; the reflexes are brisk. He stands unsteadily with his eyes shut, and he walks as if balancing a weight on his head—carefully and seeking assistance. His hands and arms are prepared for falling. His left leg drags slightly. He can raise himself from the prone position. There is no complaint of giddiness. There is rigidity of the legs and adductor spasm. The knee-jerks are exaggerated, and the left plantar reflex is certainly brisk. Ankle and knee-clonuses cannot be obtained. The muscles are in good condition.

The history of the case suggests a basic meningitis during infancy with secondary hydrocephalus. Though the history is in favour of this the condition of the skull is not that usually associated with hydrocephalus. There is a possibility that the case may be an example of hypertrophy of the brain, and it is therefore brought before the Society so that an expression of opinion may be obtained from the members.

(*Exhibited February the* 21*st*, 1902.)

40. A CASE OF HODGKIN'S DISEASE.

By A. E. SANSOM, M.D.

Phyllis A. B—, aged 8 years, was brought to the London Hospital on February the 14th, 1902, with a glandular lump in the left side of the neck and an enlarged spleen. The enlarged lymphatic glands in the neck have been noticed since the patient contracted measles, four years ago. They seem to have varied a good deal in size, and have caused her no pain. The glands composing the lump in the neck are discrete, and the skin moves freely over them.

Glands on the right side of the neck and in the groins can also be felt, but they are not very much enlarged.

On admission to the hospital the spleen practically occupied the front of the left side of the abdomen, but since the child has been in bed it has gravitated backwards.

She has a brother one year old, who has "lumps" in his neck.

She suffers from shortness of breath on exertion, and has a slight cough, but her lungs are normal. Her pulse, which is irregular, beats 100 per minute; its volume and tension are good. There is a hæmic bruit over the pulmonary area. Arterial pulsation is visible on the right side of the neck, and there are enlarged veins over the upper part of the chest.

An examination of her blood was made on March the 12th. The red corpuscles were upwards of 4,000,000 per c.mm.; the hæmoglobin amounted to 60 per cent.; the white corpuscles were 4000 per c.mm.; no abnormal cells were present.

During her stay in the hospital the temperature was practically normal. (*Exhibited March the* 21*st,* 1902.)

Discussion.

The Chairman (**Dr. Henry Ashby,** Manchester) asked what the experience of Dr. Sansom was as to prognosis in such cases. Had he ever seen a case of complete recovery? He asked Dr. Hutchison if he recommended excision of the glands in such a case. Assuming that they were tuberculous would he recommend that?

Dr. Sansom said he would like to hear from Dr. Hutchison whether he considered the condition tuberculous *ab origine* or not.

Dr. Morison asked Dr. Hutchison whether in the tuberculous cases it was usual to find a notably hypertrophied thymus gland. If there were a uniformly enlarged spleen and a greatly enlarged thymus, with a large liver and exterior glandular enlargement, was it *a priori* probable that the case was tuberculous? A number of years ago he was impressed by the fact that such a concatenation of circumstances occurred in twins. The members of the family were all more or less anæmic, and most of them grew to maturity, but none showed tuberculous symptoms. Both the twins died, the condition being what was usually called lymphadenoma. It appeared to him that the fruit from the delicate tree had resulted in splitting up; that anæmic fruit was born and broken up in the shape of lymphadenoma. That occurred before the tubercle bacillus was recognised.

Dr. Hutchison said at a previous meeting of the Society he made a somewhat strong statement about such cases, but it was not intended to be taken literally. He intended to say that so-called Hodgkin's disease was very rare, and that many cases which clinically looked like Hodgkin's disease were really an extremely chronic form of tuberculosis. That had also been stated by Continental writers, some of whom said that even if microscopical examination failed to reveal the tubercle bacillus in the glands, that did not preclude the possibility of it being tuberculous. Some even went so far as to say, that even if inoculation experiments gave negative results tuberculosis was not excluded. He had seen cases which were supposed to be true lymphadenoma, but which, *post mortem*, were found to be tuberculous. It was impossible to say during life, clinically, which form was being dealt with. Even microscopical examination was not always conclusive. He had examined the blood of the present case for Dr. Sansom, and there was nothing characteristic in it; but he did not think that was of much help, because in cases where there had been marked leucocytosis the condition was found to be tuberculous. In the last case he saw the tubercle bacillus was not found, but there was a diminution of leucocytes. It was a question whether the condition was not due to extensive sclerosis of the lymphatic glands, limiting the formation of fresh leucocytes. But in the case he mentioned he made a differential count, and the leucocytes were not diminished. In reference to tuberculous glands and the advisability of operative measures, that was a special form of tuberculosis which had a tendency to invade and progress, and many of those cases which he had looked up had been through the surgeon's hands before for the removal of a mass of glands. But the patients had returned months afterwards with a fresh mass of glands somewhere else. One of the cases was proved to be tuberculous, yet the disease had recurred. He doubted whether the prognosis was favourable even when the glands were removed. With regard to the enlargement of the liver and spleen, in undoubtedly tuberculous cases he had seen quite large spleens, with firm masses of what was called by some "monkey-tubercle," which, *post mortem*, looked like masses of suet, and very much like the condition described by Hodgkin. There were plenty of tubercle bacilli in the sections. He could not speak in regard to the thymus gland, but in that gland a sort of compensatory hypertrophy went on in many diseases in children in whom the lymphatic glands were obliterated. Even in those it was necessary to give a very guarded prognosis.

Dr. W. H. Kelson asked Dr. Sansom whether the larynx had been examined. He said that two months ago Dr. Jobson Horne brought forward some cases of Hodgkin's disease at the Laryngological Society, also pathological specimens showing ulceration of the larynx. Dr. Horne was of opinion that that ulceration was the primary lesion.

Dr. Sansom said, in reply, he believed reported recoveries could be read of. The best treatment known was the arsenical. He remembered another case which lasted a great many years, but there were tracts of adenoid tissue invading the lung and pleura. In the present case there was no such invasion, and he thought the prognosis was not of the best; he believed the larynx had not been examined, but an attempt would be made. There had been no symptoms pointing to the larynx.

41. A CASE OF AORTIC DISEASE.

By HARCOURT GERVIS, M.D.

Fred C—, aged 9 years, was admitted into Paddington-Green Children's Hospital, for shortness of breath, in September, 1898. There was a history of rheumatism contracted in July of that year.

The heart.—The apex-beat was in the sixth space, half an inch outside the nipple line. The upper border of cardiac dulness was at the junction of the fourth left costal cartilage with the sternum; there was no extension of dulness to the right of the sternum. On auscultation a double murmur was heard at the apex, presystolic and systolic in point of time, but the aortic sounds were normal. There was also heard a to-and-fro rub along the left border of the sternum, which was considered to be pericardial, and it disappeared after the application of a small blister.

The boy was sent to a convalescent home after three weeks' stay in the hospital. On leaving there at the end of another month he attended the hospital as an out-patient, and there was then heard for the first time, in addition to the murmurs at the apex, a double murmur—systolic and diastolic in point of time—over the aortic area.

The heart has remained in much the same condition ever since. The boy enjoys good general health, but has fainted suddenly on one or two occasions.

(*Exhibited March the* 21*st,* 1902.)

Discussion.

Dr. Sansom said he had briefly examined the child, and had no doubt there was aortic disease with an hypertrophied left ventricle. There were the murmurs of aortic obstruction and regurgitation over the aortic area. The signs were a little more difficult at the apex. A presystolic murmur was present, but it was terminated not by a sudden first sound but by a dull one. He could not hear anything systolic at the mitral orifice, therefore the aortic insufficiency was fairly compensated. There was no consecutive dilatation of the left ventricle. Was it a Flint murmur? Flint showed that a murmur occurring in the part of the cardiac rhythm usually occupied by the presystole might be due to aortic regurgitation and not to a mitral condition. He thought it most reasonable to conclude that the sign was produced by vibrations conducted towards the apex of the heart. In all probability the first cause was rheumatism, and the stress of the mischief had fallen on the aorta.

Dr. Morison agreed with Dr. Sansom, but thought he detected a little mitral incompetency in addition. There was no distinct evidence of costal-pericardial adhesions. He asked for an expression of opinion whether in the cases which evinced a large amount of hypertrophy with some pericardial tethering, which was made evident by auscultation and percussion and X rays, operation would not be beneficial. He inclined to the view that a certain proportion of them seemed to be operable, that the operation need not be dangerous, and if it were found to be impracticable the chest could be closed up again. There was a possibility of thus removing the drag upon the heart.

Dr. Theodore Fisher (Bristol) said that, having paid some attention to the question, he was inclined to believe that Dr. Morison was mistaken as to the pathology. The adhesions of the heart to the chest wall probably had no connection with the hypertrophy, as could be ascertained both clinically and by *post-mortem* examination. In cases of acute pericarditis which had recovered, the apex of the heart could be noticed to move considerably when the patient changed frem the lying posture to the erect posture, or on changing from resting on the right side to the left. Therefore he did not see how one could hope for a good result in such cases from surgical procedures.

Dr. Gervis, in reply, said the patient had been treated simply upon the supposition that it was a case of mitral disease with systolic and presystolic murmurs. The aortic trouble evidently came on later, after the patient returned from the convalescent home. Such a history did not accord with Dr. Sansom's suggestion.

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42. A CASE OF CONGENITAL EXOPHTHALMOS DUE TO DEFECTIVE CONFORMATION OF THE BONY ORBIT.

By G. A. SUTHERLAND, M.D.,

AND

ARNOLD LAWSON, M.D.

The patient is a little girl aged 6 years. The eyes are considerably proptosed, and in a forward direction. They can be completely covered by the lids with an effort; but the mother states that the eyes are always open during sleep. The movements of the eyes and lids are normal, as is also the fundus in each eye, whilst the refraction is hypermetropic. The face is remarkably broad and flat, the latter point being especially noticeable when it is viewed as a profile. The palate is arched, and there is a large crop of adenoids blocking up what appears to be a very shallow naso-pharynx. The child, in consequence, always keeps its mouth open, and its appearance of vacuous stolidity rather belies the mother's statement that "the child is quick and intelligent." The patient is vigorous and healthy in all other respects, and has always been a strong child. An interesting point is that the mother says that her maternal aunt was afflicted in the same way from birth. In this case the proptosis was noted immediately after birth, it being the first child, and the delivery was effected without the use of instruments. No perceptible change has taken place in the eyes since then, and in the absence of all indications of disease, and in view of the general flattening of the facial bones, there can be no doubt that abnormally shallow orbital cavities are responsible for the exophthalmos.

(*Exhibited March the* 21*st*, 1902.)

Discussion.

Mr. W. H. Jessop agreed with the causation suggested by Mr. Lawson, that the floor of the orbit was defective. He had under his care, at one of the asylums near London, a woman who showed a very great lack of development of all her bones. That was seen most in connection with the superior maxilla. Her eyes were remarkably prominent; indeed, they were almost tumbling out of their sockets, such as they were.

Dr. Cautley thought the child also had an exceptionally high palate, and that the lower jaw was badly developed, especially in the middle line. Those points tended to support the view advanced by Mr. Lawson.

Dr. Hutchison said that he saw, at the Great Ormond Street Children's Hospital, a child who was almost an exact duplicate of the present one. At first he thought it was a Mongolian with special exophthalmos. The child had a very badly developed naso-pharynx, so that the finger could scarcely be got behind the soft palate. There was also flattening of the orbit, and the eyes were directed upwards. There was a high-arched palate, and the mental defect was greater than in the present case.

The Chairman (Dr. Henry Ashby, Manchester) said he presumed it was not thought that the post-nasal adenoids caused the bony deformity. Such a theory was sometimes put forward in current surgical text-books. In the present case the deformity and the adenoids seemed to be associated.

Dr. Sutherland, in reply, thought there was no real mental defect, though there was mental hebetude and backwardness, which was probably the result of imperfect breathing owing to the existence of an abnormally small naso-pharynx which was blocked with adenoid growth. If the latter were cleared away he felt sure the mental state would definitely improve.

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43. A CASE OF INFANTILE SCURVY.

By CAMPBELL POPE, M.D.

(Introduced.)

Ivy Victoria H— was born on June the 24th, 1901. The mother was unable to suckle the child, and it was soon fed on Clay Paget's modified milk. She thrived wonderfully until August the 17th, when the parents took her on a visit to Hayling Island.

On the 19th, in the temporary absence of the mother, the housekeeper gave the child cow's milk with some whisky in it to stop it crying. It was afterwards fretful, sick, and restless at night. The maid in attendance on the child had a suppurating finger, and a crop of purulent vesicles appeared on the baby's face and hands.

On August the 23rd there was offensive diarrhœa. Diarrhœa ceased early in September, and Paget's milk was continued. After this till Christmas the child thrived well, but on January the 6th the food did not seem to satisfy her. The abdomen was large and flatulent, and potato cream was tried, and afterwards Paget's No. 2 milk. Slight bleeding of the nose began about January the 11th, but by January the 22nd the child seemed quite well, and was taken to Newhaven. The next day or two the baby seemed in pain in one leg about the knee.

On January the 24th she cut her first tooth, and on January the 28th she cut a second tooth. On January the 29th, during her mother's absence on a journey, the child was put in a damp blanket in its bassinette. On February the 1st the mother returned home; the child's legs were very painful. The urine contained oxalates, urates, and blood. Gravel or stone was suspected, but was not found by an X-rays examination or by sounding the bladder. Blood still continued in the urine. There is no doubt now about the nature of the illness. The paretic condition of the lower extremities, the screams of the child when approached for examination, the typical condition of the gums in the neighbourhood of the lower incisor teeth, together with the periosteal thickening in the left femur and tibia, and to some extent in the right tibia, mark it as one of scurvy. The infant is also decidedly rickety. (*Exhibited March the* 21*st*, 1902.)

44. SCURVY IN A RICKETY BOY AGED FIVE AND A HALF YEARS.

By GEORGE CARPENTER, M.D.

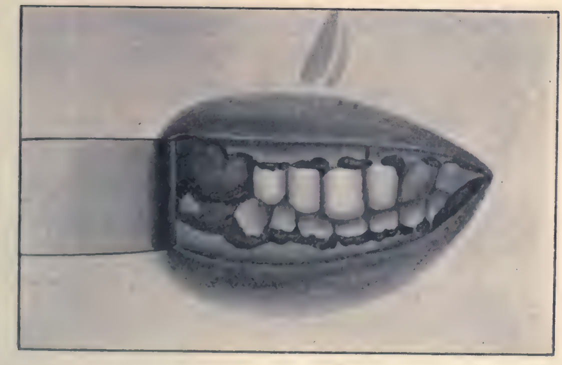
John C. M—, aged 5½ years, whom I saw for the first time on February the 24th, 1902, was breast-fed, but not weaned until eighteen months of age. When he began to walk, at twelve months old, he was found to be rickety. He was, according to his mother, always too heavy for his legs, which were gradually becoming more and more bent. About nine months ago he commenced to complain of pain in the arms and chest, and especially in the legs, and he did not like to be touched; but he attended school, however, up to within the last six months. He last walked six weeks ago, when he crawled from one chair to another, but latterly he reclined in one position, either in bed or on a chair. For the most part he was kept indoors, and had been gradually getting paler. For about a week he had cried on passing water, but no blood had been detected in the urine. He had always been constipated; the motions were free from blood.

The boy had always had peculiar ideas about his food. He would eat bread and sugar, and occasionally a little butter, but he objected to milk, though he could drink tea with milk in it. He ate fried or roast meat, but the meat had to be dry, and he would not touch gravy or meat-juice. Vegetables he always refused; they made him sick, and he had never eaten fruit of any kind. He liked suet pudding, and took milk puddings occasionally.

Apart from what has been related nothing worthy of note occurred as regards infantile disorders. His father and mother had both suffered from rheumatic fever. He had four brothers and sisters, two being older than himself, and the youngest was an infant three months old.

On examination he is found to be a well-nourished, placid, anæmic child, who remains quiet if undisturbed, but if the couch be moved or his body be approached for examination he cries out. His legs are flexed on the thighs, and the thighs on the abdomen. There is very decided tenderness all over the trunk and limbs, especially the legs. The front of the left shin has lost its sharp outline and is nodulated, and the bone is bowed. The right tibia is decidedly bowed and its free edge thickened. The shin bones can be pinched without causing him pain, when he is not paying attention. The femora are not thickened. When his attention is drawn to the fact that they are being manipulated he cries out, but not otherwise. His muscles are flabby, especially those of the legs; they are free from indurations. The tenderness and paretic condition of the lower extremities of the child, combined with his behaviour, led me to suspect scurvy and examine his mouth, although no complaints had been made of this. The mucous membrane generally is pale. At the junction of the incisor, the canine, and first temporary molar teeth with the gums there is a thin broken line of purple discoloration, which broadens out at the last-mentioned teeth, and over the second molar teeth expands into a purple epulis-like

FIG. 17.



overgrowth of the gums which almost obliterates these teeth (*vide* Fig. 17). The gums do not bleed on pressure.

The body is covered with petechiæ, mostly originating in flea-bites apparently, and there is a small bruise over the left shin.

The head is large, but not characteristic of rickets, and the anterior fontanelle is closed. The ribs are very beaded and display a prominent rosary, but in spite of this the chest is of fair shape. The wrist ends are very knobby, and the ankles and knees obviously enlarged. The long bones of the lower extremities are bent, and the curves fit into one another when the limbs are in a position of rest; the femora bend antero-posteriorly at the junction of the lower and middle thirds, and the tibiæ at the lower third. All his organs are healthy, as far as can be determined by a physical examination.

An examination of the blood shows in the fresh films typical rouleaux formation and an absence of microcytes and poikilocytes; stained films appear normal. The red blood-corpuscles number 4,733,000 per c.mm., or 94·6 per cent. The hæmoglobin is 60 per cent., and the colour index ·7. The white corpuscles number 8300 per c.mm., a ratio of 1 in 570.

The boy was admitted into the hospital under my care on the same date. With the varied diet he obtained there which he was compelled to take, coupled with orange juice, the gums quickly recovered, and by the 16th of March, some twenty-three days later, the limbs were quite free from tenderness, but he could not stand without assistance.

Remarks.—This case of characteristic scurvy of infantile type is interesting by reason of the age of the patient. Such cases mostly arise between the ages of seven and fourteen months, and usually about the ninth or tenth month, the youngest, recorded by Dr. A. Matheson, of Neilsville, Wisconsin, being but three weeks old; it was breast-fed, and ended fatally. After two years of age it is decidedly quite out of the common to meet with such an instance as that just recorded with its typical gum lesions. Although this is so I do think, and would again call attention to the fact, that some of the cases we meet with in older children suffering from *simple* hæmaturia, let me call it for the want of a better description, as also some cases of purpura, mostly unaccompanied by gum lesions and thereby passing unrecognised, are solely due to a deficient dietary, and are therefore essentially scorbutic in origin. These children are undoubtedly cured by a change to a proper mixed dietary in the hospital wards, without any adjuncts; I have seen it over and over again. Members of the Society may possibly remember last year I called attention to this view when Mr. D'Arcy Power showed a case of paroxysmal hæmaturia in a boy of nine years, which in the sequel was proved to be an undoubted case of scurvy by reason of a subsequent but tardy development of spongy gums.*

Further, so marked a case of active rickets at this period of life is unusual, and it is of interest from that point of view also, although its association with scurvy must be looked upon as an accidental one merely. Children with scurvy are not necessarily rickety, though rickets is found in about half the number of cases coming under observation. Nor are they necessarily anæmic, though in this respect they are more often so than not.

Regarding the ætiology of infantile scurvy, it is thought on all sides that the chief, if indeed not the only, cause is dietetic. In the case of this child, for instance, there can be no doubt his dietary was restricted and an unnatural one by reason of the absence of fresh vegetables or their equivalent. He did, however, take milk occasionally, in the shape of milk puddings and admixed with his tea, and he did have meat, twice a day according to his mother, though it is stated he liked his meat

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* 'Reports of The Society for the Study of Disease in Children,' vol. i, p. 158.

"dry." This child, apart from his food fads, had similar food to that of the other children at home, who remained perfectly healthy under a more varied menu.

A study of reported cases, coupled with one's own clinical experience spreading over a period of years, make it clear, to my mind, that no single article of diet is capable of producing scurvy with certainty, though it is abundantly in evidence that the various proprietary foods and milk preparations are flagrant offenders. The personal element in the case of infants and children has always to be taken into consideration, for it is a not unimportant factor in determining the onset of this disorder. Thus several infantile members of a family may be suitably reared on a diet which proves eminently scorbutic to another infantile member of that family.

Even infants at the breast, and newly born infants too, have developed scurvy, though this is not a common occurrence. Yet fresh breast milk is deemed to be eminently antiscorbutic, and it is certainly free from the ptomaines of decomposition, though in other respects it may be quite unsuitable as a food for the purpose of rearing a healthy child. Undoubtedly many articles of diet, which have been administered over long periods of time, are capable of producing scurvy in some infants, and equally undoubtedly they will not do so in the majority of others. Infantile dietetic errors among the people are the rule, but it is only exceptionally that a case of scurvy comes under observation, one unfortunate amongst the many that escape.

Thus a diet of sterilised milk, simple boiled milk, pasteurised milk, pasteurised percentage milk mixtures, peptonised milk, condensed milks, malted milks, desiccated milks, the numerous proprietary foods and cereal gruels combined with the above milk preparations, each in turn, or in combination, have produced scurvy in those prone to that disorder, though harmless in that respect to others.

According to Rosenthal, people amongst whom he practises have a method of feeding their children on Zwieback from very early life, a food similar to toasted sweet bread, nothing else, not even milk for a time, and these children after three or four months of this diet show signs of scurvy.

Further, it has been proved, in a considerable number of cases, that the addition of orange juice, or fruit juice, or beef juice, to some of the above dietaries which have been proved scorbutic, has been quite sufficient to cure the complaint.

On the other hand, there is the fact that antiscorbutic articles, in the shape of potatoes and bananas, have been taken where milk was seldom drunk or meat eaten, but were not successful in the face of so defective a dietary in warding off the disease.

In the case of some infants a mere change of diet, and that not always *primâ facie* an ideal one, and without beef juice or fruit juice, has proved successful in bringing about a cure.

The judicious combination, however, of fruit juice and a rational dietary must be looked upon as a specific, and the rapidity of the cure in such cases is most striking.

Does the view of Torup of Christiania, that scurvy is due to ptomaine poisoning from tainted meat, and the fact that fresh vegetables and lime juice form a part of the dietary is not sufficient to prevent or cure scurvy, satisfactorily explain cases of infantile scurvy? It is a view which has much to recommend it, for it has been fortified by Arctic experiences, and has, moreover, been confirmed by the experiments of Jackson and Vaughan Harley on monkeys.

My own clinical experience of infantile scurvy, combined with a study of the histories of numerous fully reported cases scattered through literature, do not favour the acceptance of Torup's view on adult scurvy in explanation of its ætiology.

It would appear in infantile scurvy that at present we can go no further than to say the fault mainly lies in the diet, but not from a ptomaine point of view; and the more the food departs from what is natural, and the more prolonged is the use of such a faulty food, the more likely is it to produce scurvy. But for reasons already given there must be a personal factor in these cases. The diet prescribed may not be appropriate for the particular child for which it has been designed. Certain foods are specially obnoxious to certain children, and of these harmful foods the proprietary foods hold the pride of place. (*Read March the* 21*st*, 1902.)

Discussion.

Dr. Sutherland said a case of hæmaturia was shown before the Society last year in which the presence of a renal calculus was thought possible. A surgeon had operated upon it, but found nothing, and the case afterwards proved to be one of scurvy. In that case, as in that of Dr. Campbell Pope's, he thought the hæmaturia was due to scurvy, but the presence of a large amount of oxalate of lime crystals or uric acid would probably also occasion pain, which seemed to have been present in this case also. He had not had experience of the particular food which this child had been given, but the makers of it supplied with each bottle directions that after a certain age the child should be given meat juice or orange juice. He did not regard that as a satisfactory way of bringing up children. Foods of that kind were injurious to children, and in the case of a dangerous food it was not satisfactory to require the administration of an antidote. It was much better to select a food which would suffice for every healthy child. Patent and proprietary foods had their value, and that was recognised in illness, but they should not be used systematically for the bringing up of healthy infants. The child shown by Dr. Campbell Pope was not only scorbutic but very rickety, and the deformity in its chest wall was very striking, and the beading of the ribs greater than usual. The condition of the radii confirmed his view. The case narrated by Dr. Carpenter was very interesting at that age. He (Dr. Sutherland) saw one case, aged five years, before infantile scurvy was recognised by the profession. It died in the hospital from pneumonia, but there was extreme tenderness about the limbs, and subperiosteal extravasation of blood. He asked whether Dr. Carpenter thought the prognosis in his case was grave. His own experience was that when a certain stage had been reached in the scurvy, and the anæmia was pronounced, it was a serious affection. He had had several cases which died, though they were under antiscorbutic treatment, and after all active scorbutus had passed away. Similar cases which died under treatment had been published.

Dr. C. W. Chapman laid great stress on the necessity of the milk being quite fresh. He had treated a similar case to Dr. Pope's, which had been fed upon preserved milk. The legs were swollen, and he thought the case was one of rheumatism. It rapidly got well under fresh milk and orange juice. The profession was much to blame for recommending some of the foods which were used. If practitioners would determine to order fresh milk there would not be so many rickety children.

Dr. Cautley suggested that the title of Dr. Carpenter's communication should be amended to "A Case of Scurvy in a Boy of Five affected with Rickets." He thought the scurvy had no association with the rickets, though both of them were probably due to unsuitable food, and, as Dr. Chapman had pointed out, the unsuitability consisted in want of freshness. There was some virtue in fresh milk which was not operative when the milk had been cooked. It took a long time to develop scurvy under suitable conditions of feeding even if the milk were boiled or sterilised. He remembered one case which was brought up on Pasteurised milk, and it took ten to twelve months for the scorbutic symptoms to develop. These were very well-marked when he saw the child, but by altering the diet to fresh milk no symptoms remained in a week. In another case, in which he deemed it advisable for the child to be fed on sterilised milk, five or six months elapsed before the development of symptoms. When it was brought with scurvy it was sufficient to bring the milk merely to the boil to cause the scorbutic symptoms to subside. He agreed with the remarks of Dr. Sutherland on the subject of the proprietary foods, and that they were not necessary for the bringing up of healthy children. Any healthy child could be brought up on cow's milk.

Dr. Theodore Fisher (Bristol), in reference to the mortality in cases of scurvy, suggested it as possible that the cases which ended fatally might have acquired a septicæmia, to which the scorbutic condition strongly predisposed. In a fatal case of the kind which he had examined two months ago he found a staphylococcus infection.

The Chairman (**Dr. Henry Ashby**, Manchester) said, in reference to the remarks on special foods, that a large illustrated advertisement could be seen at most railway stations, quoting the words of a medical baronet, "Admirably adapted to the wants of infants." If there were any friends of that baronet present he hoped they would give him a nudge when they had the opportunity.

Dr. Campbell Pope (introduced), in reply, agreed with the remarks of Dr. Sutherland and Dr. Cautley as to feeding, but medical men had been trying to steer between Scylla and

Charybdis, the Scylla being diphtheria, typhoid, and tubercle, and Charybdis being rickets. How was one to steer clear of the difficulties? By bringing the milk to the boil possibly the friendly germs in it would be destroyed.

Dr. George Carpenter, in reply, said two of his cases had died in spite of treatment, but they had the condition severely, and both of them had separation of the epiphyses of the lower extremities. He thought the prognosis in this case was good. Cases of recurrence of scurvy had been placed on record in which recovery occurred, and one or two had been reported which had died. With regard to the freshness of the food, one reported case had been reared on fresh cow's milk. Several cases had been recorded of scurvy in breast-fed infants, and surely no food could be fresher than that. Apart from that he quite agreed as to the need of freshness in milk, and that infants required something better than proprietary foods, which were most injurious. With regard to the title of the paper the printed agenda was erroneous. His own heading was "Scurvy in a Rickety Boy aged Five and a Half Years," and he had read that title to the meeting. He had specially called attention to the fact in his paper that scurvy and rickets had nothing to do with one another, but they were probably both due to improper feeding.

Postscript by Dr. Campbell Pope (April the 17th, 1902). —The child is now quite well, and has been fed mainly on unsterilised milk and barley-water.

45. A CASE OF INTUSSUSCEPTION OF MECKEL'S DIVERTICULUM.

By W. P. MONTGOMERY, B.S.

The patient, a boy aged 2½ years, was admitted to the Manchester Children's Hospital on January the 2nd, 1902. He had always been a healthy child. Forty-eight hours before admission he was seized with griping abdominal pain, vomiting, and diarrhœa. Twenty-four hours later a quantity of blood appeared in the stools. This hæmorrhage was not repeated.

On admission a tumour could be felt in the right hypochondrium. The child was much collapsed. At the operation an ileo-colic intussusception was found, and with difficulty reduced, two feet of the ileum being invaginated. The apex of the intussusceptum was found to be the inverted Meckel's diverticulum. The inversion of this could not be completely reduced, so a suture was passed through the base of the process and tied in order to prevent any subsequent relapse. The child was too ill to allow of the proper course in such cases, the resection of the process. Death took place nine hours later.

At the autopsy, made two days after death, the specimen shown was removed. The diverticulum was situate two feet above the ileo-cæcal valve.

The condition is a very rare one. Gibson, in his collection of 239 cases of intussusception in 1897, does not mention a case. Treves, in the 1884 edition of his book, mentions one case in Guy's Hospital Museum; and in the last edition, one in the museum of the College of Surgeons. He also quotes a case by Adams. Erdman's case in 1898, Boldt's in 1900, and Wainwright's in the 'Annals of Surgery,' January, 1902, are apparently the only others on record. (*Read March the* 21*st*, 1902.)

46. A SPECIMEN OF CONGENITAL HEART DISEASE SHOWING VEGETATIONS ON THE PULMONARY AND AORTIC VALVES.

BY THEODORE FISHER, M.D.

THE specimen shown was the heart of a female infant, aged 4 months, which presented an opening between the aorta and pulmonary artery in an unusual position. This opening, which was sufficiently large to admit the tip of the little finger, was situated mainly above the level of the aortic and pulmonary valves, but extended downwards into the upper part of the septum ventriculorum,—that is to say, associated with the common defect of a patent septum ventriculorum was the rare lesion of a communication between the aorta and pulmonary artery below their point of exit from the pericardium. Only two segments of the pulmonary valve were present, but the aortic valve possessed the normal three segments. An interesting feature was the presence of large but organised vegetations on the pulmonary and aortic valves. The infant was much wasted. There was a left hare-lip, and the rectum and vagina had possessed only one aperture, but this had been operated upon successfully by Mr. Paul Bush.

The child was the sixth; the others were living and well. The mother had never suffered from rheumatism, but during her pregnancy bad smells had been present in the house, and the drains were discovered to be defective. After the birth of the child these drains were repaired by the landlord.

The possibility of the endocarditis having been infective in nature is suggested not only by reason of the size of the vegetations, but by the history of the case. Infective endocarditis when occurring in post-natal life often shows extensive signs of healing, and possibly infective endocarditis in the fœtus may completely heal.

(*Exhibited March the* 21*st*, 1902.)

47. PETECHIÆ IN THE STOMACH IN DIPHTHERIA.

By THEODORE FISHER, M.D.

The stomach of a female child, aged 4 years, was shown which had died of pharyngeal diphtheria. It was thickly studded with small circular petechiæ, which ended abruptly at the pylorus and at the œsophageal end. No petechiæ were present elsewhere, except in the large intestine, where a few surrounded solitary follicles which could be seen as white spots in the centre. Their connection with solitary follicles in the stomach was not obvious. The small intestine was quite free from these small hæmorrhages, and no petechiæ were present on the serous membranes. The child had suffered from severe vomiting during the last two days of its life. The vomit gave evidence, from the onset, of the presence of blood.

(*Exhibited March the* 21*st*, 1902.)

48. A CASE OF PURULENT PERITONITIS ASSOCIATED WITH EMPYEMA (POLYORROMENITIS); RECOVERY.

By HENRY ASHBY, M.D.

It is by no means uncommon to find multiple serous inflammations at the autopsies of those who have died of acute disease of a septic character. In such cases there has been an infection of the organs by the streptococcus, the pneumococcus, etc., and the cocci have been carried from one organ to another by the blood-stream. The only wonder is that it does not occur oftener. Among the commonest associations may be noted a double pleuro-pneumonia, or pleurisy, a pleuro-pericarditis, pleuro-pneumonia and meningitis, peritonitis and pleuro-pneumonia. Such cases have been dignified by the name of "polyorromenitis" or "polyserositis." * It must, however, be obvious to every one that these cases differ widely from one another as regards the infection and the serous membranes affected, and only confusion can result from including such diverse cases under a common name.

The following case is, I think, of interest as illustrating one form of combined serous inflammation associated with the *Micrococcus tetragenus*, and ending in recovery in spite of the suppurating peritonitis which occurred.

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* See Dr. Fred. Taylor, 'Brit. Med. Journ.,' 1900, ii, p. 1693.

The patient was a girl, aged 8½ years, who was delicate and had suffered much from repeated bronchitic attacks.

On September the 11th, 1901, she was seized with vomiting, diarrhœa, and fever; there was also some abdominal pain. The attack was attributed by her friends to her having taken some tinned salmon the evening before, but as no one else who had taken the same dish had suffered, it is more than likely the salmon had nothing to do with her illness. When seen next day by her doctor (Dr. Senior) the temperature was 103° F., the vomiting incessant, and the stools were liquid and frequent; there was also cough and expectoration. The vomiting ceased in a few days, but the diarrhœa and fever continued for a week; the temperature ranged from 103° F. to 104° F.; pain was complained of in the abdomen and left side of the chest. On the 21st, when I saw her, she had been in great pain, referred to the abdomen; at times this had been very distressing: there was tenderness over the abdomen, and much tympanites. There were also signs of an extensive consolidation of the lower two-thirds of the left lung, and a patch of consolidation at the right base. The respirations were rapid; the temperature 101° F., the pulse 140 to the minute. Both Dr. Senior and myself thought the prognosis extremely bad on account of the complication of peritonitis with double pneumonia. On the other hand, when not in pain she was bright, and there were no signs of collapse.

The next twelve days brought no improvement; the temperature continued elevated, ranging towards the end of this period from 99° F. to 103° F., and when I saw her again there were all the signs of large collections of fluid in the peritoneal and left pleural cavities. The abdomen was much distended, enlarged veins were visible on the surface, and there was a zone of redness around the umbilicus. The flanks were bulging and dull, and a very marked fluid thrill was readily obtainable. The patch of pneumonia on the right side had disappeared, but the left

Fig. 1.

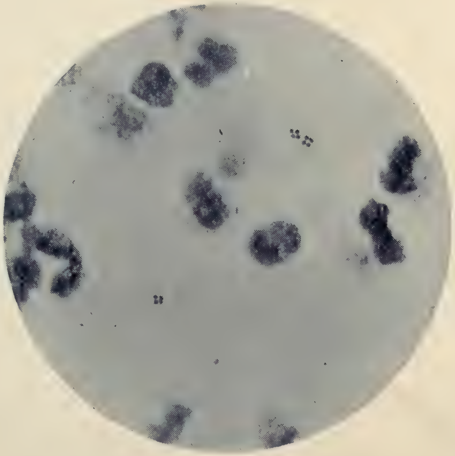
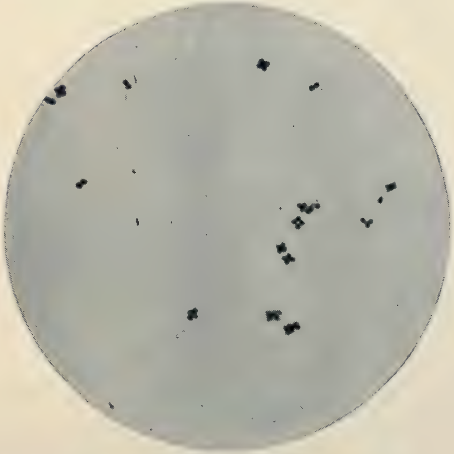


Fig. 2.



Dr. Henry Ashby's Case of Purulent Peritonitis associated with Empyema.

Fig. 1.—Peritoneal pus showing *Micrococcus tetragenus*. Capsules not visible in the photograph. Leucocytes contain cocci. × 850.
Fig. 2.—Agar cultivation of *Micrococci tetragenus* from empyema. × 850.

Adlard & Son, Imp.

side of the chest was very dull on percussion, and the heart was displaced to the right.

An operation was decided on at once, and next day Mr. G. A. Wright evacuated forty ounces of pus from the abdomen by inserting a tube at the umbilicus, but before his arrival a slight oozing of pus had taken place at this spot from the pus having found its way through. At the same time he excised a piece of rib from the left side of the chest and drained the empyema.

Much pus drained away during the next few days from the abdomen, but ceased to flow in a few days, and on the fifth day the tube was removed. The empyema gave more trouble, but finally a good recovery was made. The temperature fell on the day of the operation, and remained normal after with slight exception, due to the empyema not draining perfectly.

Mr. W. J. S. Bythell was good enough to make a thorough examination of the pus from both cavities. He reports:

A. *Peritoneum.*—The pus is thick, viscid, pale, greyish green, and inodorous. Microscopically it is very rich in cells, and there is a moderate amount of fibrin. It is very crowded with cocci, all of which are spherical and capsulated, and arranged mostly as tetrads. There are no pneumococci, or tubercle bacilli. There is a fair amount of phagocytosis (see Plate V).

The cocci stained well by Gram's method; some of the capsules were faintly stained by eosin.

Vigorous *cultures* of the *Micrococcus tetragenus* appeared on agar and gelatine, without liquefaction of the latter.

B. *Empyema.*—The pus is thinner than the above, greyish in colour, and inodorous, rapidly forming an upper layer of cloudy serum.

Microscopically it contains fewer pus corpuscles, and a fairly large amount of fibrin. It also contains a comparatively small number of cocci in the shape of (*a*) typically lanceolated pneumococci, with (*b*) a few tetrads composed of spherical cocci. Both forms are distinctly capsulated, and stain well by Gram's method. There is a good deal of phagocytosis. There are no tubercle or other bacilli.

Cultures are less vigorous than the peritoneal; there are colonies of both pneumococci and *Micrococcus tetragenus* (see Plate V).

The case was in many respects a remarkable one. A delicate girl is seized suddenly with a concurrent peritonitis and double pneumonia, followed by suppuration; recovery ensues almost without a bad symptom. The pus from the abdominal cavity was found swarming with cocci, and yet the general symptoms were comparatively mild. What was the exact significance of the presence of the *Micrococcus tetragenus* in the pus of the peritoneum and in that from the pleural cavity I am unable to say; in this case, at least, it was associated with mild symptoms.

In what way were the empyema and the peritonitis related? Here again is a difficulty. As far as the time of onset is concerned they apparently commenced together, and possibly owed their origin to the same infection, namely, the *Micrococcus tetragenus,* though in the empyemic pus Mr. Bythell also found the pneumococcus.

But little is known, I believe, about the part played by the *Micrococcus tetragenus* in suppurations; but it seems occasionally to be found in empyemata and also superficial abscesses. (*Read March the* 21*st*, 1902.)

Discussion.

Dr. Sansom thanked Dr. Ashby for his most interesting paper. The author's suggestion to inquire into the bacteriology of disease-states, and the hopes of inducing artificial immunity, were very encouraging. There were evidently many micro-organisms capable of causing suppuration. He could look back on the time when the suggestion about micro-organisms was met by the challenge, "Show us your organisms." That had been gradually met. Everything showed the necessity of going to the root of matters.

Dr. Theodore Fisher (Bristol) said he had seen in the *post-mortem* room one or two cases of pneumococcal peritonitis. One case was pneumococcal peritonitis associated with pneumonia. In the other cases, although an encapsuled diplococcus was found, streptococci were also present. With regard to prognosis, he thought Dr. Ashby would agree that in children definite pneumococcal infections of the lung were not by any means very serious cases, and the majority of them ended favourably. By definite pneumococcal infection he meant lobar pneumonia. Possibly in cases of so-called broncho-pneumonia the organism at the bottom of it was not the pneumococcus at all. Those were more serious cases, and about half such died. In the one case there was clinical evidence of extensive consolidation of one lobe, and the child got well. On the other hand, there were simply small patches, no larger than tubercles, scattered through both lungs, yet they ended fatally. It was unreasonable to conclude that the first class of case was due to the same organism as in the second. If the diplococcus found in pneumococcal peritonitis were the true diplococcus pneumoniæ it seemed to him that the peritonitis should by no means be necessarily fatal.

Dr. David Nabarro said that last year at the Evelina Hospital, out of eighty *post-mortems* four showed purulent pericarditis associated with empyema, and in two purulent peritonitis was present also. In all of them the pneumococcus was present. The empyema was on the left side in all the cases. He thought the general infection was by direct spread from the empyema to the pericardium, and through the diaphragm into the peritoneal cavity. All the cases were about two years old. There was no gastric ulcer, or any sign of localised peritonitis, but the lymph and pus had gravitated to the pelvis.

Dr. H. J. Simson suggested that the history in Dr. Ashby's case pointed to a gastric origin. The primary symptoms might have been due to some abdominal cause; if so, the absence of the pneumococcus would be explained. The question was whether the pneumonia was not spread from the peritoneum, and the empyema the secondary trouble. He had seen a child aged eight years with a gastric ulcer, and an abscess formed between the stomach and the diaphragm and spread to the pleura. The child next developed suppurative pericarditis and died. The nature of the case was only cleared up *post mortem*. Was the present case one of abscess round the common bile-duct, from which infection spread and gave rise to pneumonia followed by an empyema?

Dr. E. W. Goodall was of the opinion that cases like that described by Dr. Ashby should be separated from those mentioned by other speakers in which a definite source of infection was found, such as a gastric ulcer or a subdiaphragmatic abscess. In these latter cases the local lesion might serve as the point of entrance of almost any infection. The question of diagnosis was both interesting and important. He had seen two or three cases of combined pleural and peritoneal inflammation which had been sent into a fever hospital as typhoid fever. In the last case he had seen there was suppurative general peritonitis; the case was remarkable because pus had made its escape not only, as in Dr. Ashby's case, through the umbilicus, but also through the stomach. The patient vomited a quantity of pus. It was clear from the *post-mortem* appearances that the pus had come from the peritoneal cavity into the stomach, and that it was not a case of a gastric ulcer perforating.

The Chairman (Dr. Henry Ashby, Manchester), in reply, said there were many pneumonias of a gastric type, which would throw the doctor off the scent. Signs of pneumonia would sometimes follow gastro-intestinal symptoms. He had been anxious to know what was the meaning of the tetrad; did it make for malignity or did it tend to benignity? Apparently the condition of things in his case was benign rather than malign.

49. CYSTIC LYMPHANGIOMA OF THE NECK UNDERGOING SPONTANEOUS CURE.

By DAN McKENZIE, M.D.

A FEMALE baby, aged 11 months, was found to have immediately after birth a tense cystic tumour in the posterior triangle of the neck. It occupied a position about halfway between the mastoid process and the clavicle, and was about the size of a small orange. The cyst was so firm that the finger could not be pushed through to the underlying solid tissues. There was a faint suggestion of blueness of the skin over the tumour. Three weeks later the cyst had become so flaccid that its floor, and to some extent its deep relations, could be made out. It was found to lead by an infundibuliform opening under the posterior edge of the sterno-mastoid.

From the cystic nature of this congenital tumour, together with the existence of deep attachments, the diagnosis of cystic lymphangioma—hygroma—was made. As there seemed to be some tendency to softening, active treatment was withheld in the hope that a natural cure might result—a not uncommon event in this class of tumour, for which reason it is said (Bland-Sutton) that one rarely comes across these cysts after puberty.

The state of matters now seems to indicate that the hope will be realised. In most positions in which the neck is held the tumour is barely visible. The skin over the affected part is wrinkled.

On palpating the tumour now one can make out, in addition to the original cyst, two small, hard, oval bodies, lying in close relationship to the deep wall. These, I suppose, are two small cysts of the same nature as the large one. Cystic lymphangioma of the neck is often multilocular. There is no macroglossia.

(*Exhibited April the* 18*th*, 1902.)

Discussion.

Mr. Whitelocke (Oxford) said he was aware that Bland-Sutton mentioned these tumours were seldom found after puberty, but they did occur after that time. In one case he had seen the subject was aged twenty-seven years. In the present case the tumour seemed likely to disappear entirely.

50. PERONEAL TYPE OF MUSCULAR DYSTROPHY.

By EDMUND CAUTLEY, M.D.

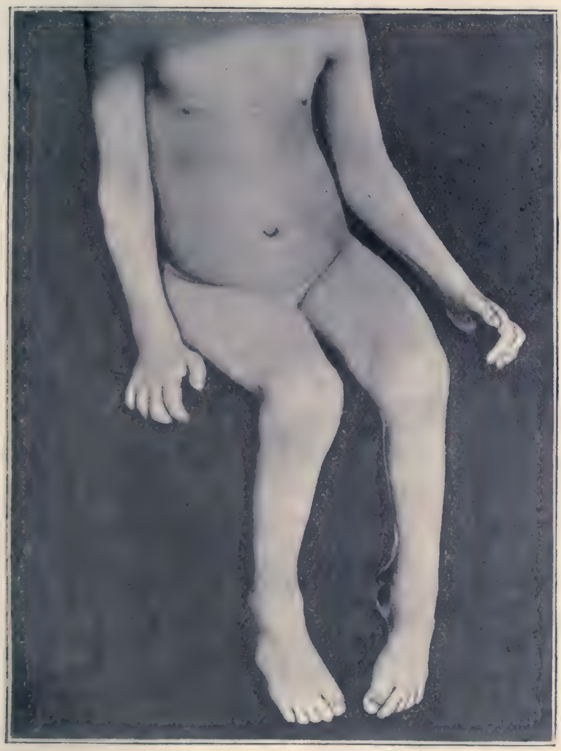
Edith H—, aged 7 years, is the fourth of a family of six children, of whom the third and fifth died under the age of one year. The parents are healthy. A brother and sister of the mother are epileptic. There is no other history of nervous or muscular disease.

The patient was a fine fat baby. She recovered satisfactorily from an attack of measles at six months old, but did not walk until three years of age. The hands never seemed quite the same as those of a normal infant, and were used differently, objects being picked up with the whole hand rather than with the finger and thumb. On walking the child supported herself on the outer sides of the feet. Abnormality in the shape of the feet was noticed at eighteen months of age, there being some degree of talipes, and the toes were unduly flexed. From the time the child began to walk the legs became weaker and thinner. The hands began to lose power at five years old, becoming more useless, thinner, and gradually "claw-like." This deformity of the hands has now been present for over two years. Her present condition is that of a normal child in every respect but two. She is not quite as mentally developed as she ought to be, but the deficiency is not more than can be explained by the fact that she has not been to school. Her muscular system shows a remarkable degree of paralysis and atrophy with a characteristic distribution. Secondary to the muscular dystrophy are the deformities of the feet, talipes equino-cavus on the left side, and to a less extent on the right. The left tendo Achillis is much contracted. The right foot can be brought to a right angle with the leg. The co-ordinating power is good, except for the presence of the muscular weakness, which renders the finer movements difficult. The knee-jerks are absent, and the plantar reflexes barely obtainable. No fibrillary tremors have been noted.

Muscular system.—The muscles of the trunk, shoulders, and upper arms are normal. The extensors of the fore-arms are wasted, and there is great wasting of all the muscles of the hands. Movements of pronation and supination are normal. There is very slight power of abduction and adduction of the fingers from the middle line. The thighs are wasted, the wasting being most marked in the vastus internus muscle and on the left side. Movements of abduction and adduction are normal. There is very great wasting of the muscles below the knee, especially the peronei and the extensors. The intrinsic muscles of the feet are also atrophied (*vide* Fig. 18).

Electrical reactions.—Dr. E. Farquhar Buzzard kindly made an exhaustive examination of the muscles, and reported as follows:—The lack of electrical response corresponds generally with the want of voluntary power. Nowhere is there sign of true reaction of degeneration. In the upper extremities all the muscles about the shoulder and upper arm respond well to both currents. In the forearms the responses are good, except in the supinator longus muscle and extensors of the wrist, which do not react as well as normally. There is no response to either current in the intrinsic muscles of the hands. The glutei and the tensor fasciæ femoris muscles react but feebly, and a feeble reaction, with a strong current, is at times obtainable in the hamstrings and the right vastus internus muscles. There is no response to either faradism or galvanism in the other muscles of the thigh, or in any muscles below the knees.

FIG. 18.



There is, I think, no doubt that this is a case of that variety of muscular dystrophy which was fully described by Tooth, in 1886, under the name of "The Peroneal Type of Muscular Atrophy," and by Charcot and Marie in the same year. It is an early case, for usually the disease begins in the second half of childhood. It is of interest, too, as an isolated case in a female. Males are affected in the proportion of two to one. The disease has been traced through five generations by Herringham,* and in the male members of the family only. Like pseudohypertrophy it especially affects males, and is transmitted by females. It is typical in its mode of onset and course. Usually it begins in the intrinsic muscles of the feet, the extensors of the toes, or the peronei, or in several of these muscles at the same time. It spreads to the other muscles of the leg and thigh, especially affecting the vastus internus. It spreads to the intrinsic muscles of the hands causing atrophy of the thenar and hypothenar eminences, and of the interossei producing the "claw-like" hand. This peculiar deformity is an extremely rare condition in early life from any other cause. The atrophy and paralysis are generally symmetrical.

Pathologically it is not improbable that some of the cases are due to a congenital tendency to muscle or nerve degeneration, which may be excited by toxins setting up a neuritis, or acting directly on nerve or muscle. A chronic peripheral neuritis has been found in two cases. Possibly some are due to a congenital tendency to premature failure of nutrition in the peripheral nerve-fibres, apart from any definite exciting cause. There is a marked tendency for the disease to follow acute specific fevers, especially measles. I take the view that in this case the affection was started by the attack of measles.

(*Exhibited April the* 18*th*, 1902.)

Discussion.

The Chairman (Dr. Leonard Guthrie) thought there could not be any doubt that the condition was one of myopathy. Dr. Cautley had referred to it as the disease originally described by

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* 'Brain,' 1888.

Howard Tooth as a type of muscular atrophy, but his impression was that Tooth's cases were ascribed to a certain amount of peripheral neuritis, whereas the sensation in the present case seemed normal and there was no pain. With regard to family history, often that could not be obtained, but in some of the cases close investigation showed that some other members of the family in days gone by had suffered from some mild form of complaint which had not attracted attention. If contraction of the tendon prevented the child from walking, perhaps that tendon might be divided with some advantage, but he would not recommend any other surgical measure.

Dr. Fletcher Beach said, with regard to the talipes, the late Mr. Cotterill, the surgeon who was at the West-end Hospital for Nervous Diseases, was in the habit of excising the outer part of the foot, or whatever other part was required according to the nature of the disability, so as to bring the foot at right angles with the leg. The effect was to make a permanent flat foot, but Mr. Cotterill thought that preferable to the original deformity. He did not know whether that procedure would be good in the present case, because so many muscles seemed to be involved since the peronei were first affected. Some time ago a child was sent into the hospital under his care with supposed peroneal wasting; but on examination there was found to be nothing of the sort. The part was very tender to the touch, and the case turned out to be one of sciatic neuritis, which got well under treatment. Possibly massage in the present case might check the rapidity of the wasting of muscle.

Mr. H. J. Curtis said, with regard to treatment of the tendon for the pes cavus with equinus which resulted from the paralytic condition of the calf muscles, to do tenotomy of the tendo Achillis would not improve the walking power. The treatment under the circumstances he thought best, would be to lengthen the tendon.

Dr. Dawson Williams asked whether Dr. Cautley was prepared to accept the theory of peripheral neuritis in his case, because that was of considerable interest from the point of view of ætiology. He was glad to hear Dr. Cautley lay some stress on the occurrence of measles in the history at some time not very long before the onset of the paralytic symptoms. If he were prepared to accept the possibility of a peripheral lesion, then the ætiology was to some extent cleared up. It was much more easy to accept the doctrine that measles could produce peripheral neuritis of a temporary character than that there was a central or purely muscular lesion which could be produced by a toxin, because, as far as he knew, central lesions produced by toxins were very uncommon, if not unknown.

Dr. Sutherland thought the muscular weakness below the knee was so marked in the child that surgical treatment would not do any good. The foot could perhaps be straightened, but the walking power would not be improved. The case was different in spastic paraplegia.

Dr. Cautley, in reply, thought his case certainly belonged to the variety described by Tooth. Under the name "peroneal atrophy" several different affections were included. He thought some of the cases were due to primary nerve degeneration or to primary muscle degeneration, set up by toxins or secondary to neuritis. He had laid stress on the possibility of the present cases following on measles; that there was a degeneration of nerve following on the measles. Dr. Dawson Williams had published some very interesting cases of neuritis following measles, but there was some additional factor in the majority of the cases of the kind he had shown. There was a great tendency for the affection to run in families, so that there must be some congenital weakness; and in such cases the affection might occur without any traceable exciting cause. He thought no benefit was obtainable in the case from any form of treatment whatever, whether massage, electricity, tenotomy, or tendon-grafting. The sole advantage that could be gained by lengthening the tendo Achillis was in allowing the foot to be flat on the ground.

51. TUBERCLE OF THE CHOROID.

By SYDNEY STEPHENSON, C.M.

Alice P—, aged 8 years, an inmate of the Ophthalmic School, Hanwell, W., was examined on April the 5th, 1902.

1. *Family history.*—Unknown.

2. *General condition.*—The child looks well. Her height is 43 inches, and her weight 41½ lbs. The pulse-rate is 104 to the minute; the respirations are 18 to the minute; the temperature is 98·6° F. The spleen is not palpable. The liver is not enlarged. The apex of the left lung has a higher pitched percussion note than that of the other side; there is also diminished expansion, increased vocal resonance, and doubtful bronchial breathing. No accompaniments can be made out. The lung signs are not very marked.

3. *Condition of the eyes.*—Lying ¼th of a papilla's diameter below the right optic disc, is a somewhat semicircular patch of glistening white hue, measuring perhaps

10 mm. by 5 mm. It is crossed by two retinal arteries and a like number of retinal veins. It has a small and ill-defined collection of pigment at its upper and outer side. Situated a little lower down, separated from the foregoing lesion by an area of normal choroid, is an oval black spot some 5 mm. in length (*vide* Plate VI, between pages 206 and 207). This probably represents a "satellite tubercle."

(*Exhibited April the* 18*th*, 1902.)

Discussion.

The Chairman (Dr. Leonard Guthrie) inquired what was the prognosis of tubercle of the choroid in Mr. Stephenson's experience. He (Dr. Guthrie) had always regarded it as particularly grave.

Dr. George Carpenter said that no doubt the members who were present at the Liverpool meeting of the Society last year would remember seeing numerous water-colour drawings of cases of tuberculosis of the choroid which Mr. Stephenson and he then exhibited. The present case brought before the Society was one associated with a tuberculous lesion elsewhere, viz. disease of the apex of the left lung. One case shown at Liverpool was that of a child, with a very similar choroidal tubercle to that in the present case, who died suddenly, and the only lesion detected *post mortem* was chronic tuberculosis of the apex of one lung. One of the cases demonstrated at Liverpool, with somewhat similar fundal changes to the case shown, had a tuberculous tumour of the brain. With regard to prognosis, he thought it was usually that of the disease with which they were associated, viz. in tuberculous meningitis and general tuberculosis fatal. In a case of coarctation of the aorta, in a child of five years old * under his care, he had found the fundus oculi healthy when examining for alterations in the blood-vessels. Ten days later, owing to a hectic temperature and steady emaciation, a further search was made, with the result that several miliary tubercles were detected in the fundus together with slight papillitis. Here the choroidal tubercles, the optic papillitis, and the steady physical retrogression appeared to develop side by side, and the issue was rapidly fatal from tuberculous meningitis and probably general tuberculosis, though unfortunately the child had then disappeared from his view. On the other hand, Dr. Sansom at the Liverpool meeting mentioned a case in which he was called in consultation to see a child who was suffering from cerebral symptoms, and when that observer detected several choroidal tubercles in the fundus oculi he was no longer in doubt as to its grave nature, and gave a fatal prognosis. But Dr. Sansom was wrong, as the child was again accidentally seen by him some twelve months after, quite recovered, though he had not unfortunately the opportunity of re-examining it. His experience was not so unfortunate as that, and in cases associated with the previously mentioned diseases where he had detected choroidal tubercles the end was invariably fatal within a short period of time. Dr. Sansom's experience, however, was a valuable one, and should be borne in mind. In chronic cases, cases associated with chronic tuberculous lesions elsewhere, such as phthisis, tuberculous peritonitis, tuberculous joints, tuberculous brain tumours, and spinal caries, the prognosis was much that of the disease with which they were associated, with the reservation that such were not unlikely to develop, if not at the time at some future date, miliary tuberculosis. But in that respect he did not think they differed from similar cases without the eye lesions. He had watched one such case in a child with spinal caries for twelve months, during which time the tubercle, which was a fawn-coloured body, developed pigment in its centre in the form of a ring. Subsequently the ring formation of pigment dispersed, and the face appearance of the tubercle was altered during the period of observation. Next whooping-cough supervened, and the child died of general tuberculosis. In obsolescent cases the detection of choroidal tubercle should be considered a valuable introduction to the constitutional taint lurking there, which may not be at all obvious, and which, when found, can be guarded against. Further, the detection of choroidal tubercle may assist in forming a correct opinion in a not very obvious case, as had happened to the speaker on several occasions, or lend aid in arriving at a conclusion as to the nature of the disease under observation, such as the character of a cerebral tumour. Often as a valuable aid to diagnosis, therefore, there can be no question as to the assistance they render when detected. There is no doubt also the appearance of choroidal tubercles often antedates slightly and heralds the more obvious signs of meningeal and general tuberculosis, which at that time may be reviewed by the mind as possibilities merely. When discovered a prognosis should not be founded on them, but rather on the condition of the patient. It did not follow, in acute and chronic cases of tuberculosis, because there were tubercles in the fundus oculi tubercles were necessarily in the meninges or disseminated generally. On the contrary, *post-mortem* evidence had shown him that the disease might be localised to the choroid and one other spot, such as the lung for example, so that the founding of a fatal prognosis on their detection in the fundus oculi was unwarranted and unwise. Furthermore, reviewed from the clinical aspect, choroidal tubercles may remain quiescent for years, and the patient keeps well and hearty during that time.

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* 'Archives of Pediatrics,' vol. ii, p. 330, 1896.

Mr. H. J. Curtis confirmed Dr. Carpenter's observations.

A case of Mr. Stephenson's with tubercle of the choroid which he had recorded in his book was still in the hospital when he (Mr. Curtis) had charge of the beds during the absence of the surgeon. A child was brought with tuberculous disease of the knee, and he excised the joint as cleanly as possible, but recurrence occurred in about ten days. He reopened the joint and found a complete recurrence of tubercle, not a septic invasion, so that the joint looked as if it had not been dealt with before. He found it necessary to amputate at the middle of the thigh, and the child recovered, and was well three months later.

Mr. Stephenson, in reply, said the question of prognosis was a difficult one to answer. He was familiar with two classes of case: one of these being in which the tubercle was recent, and in which the patient in most cases died quickly. He showed such a case at an earlier meeting of the Society this session, in reference to which it was a question whether the child would not die while being shown. It did die two days later from generalised tuberculosis. He had watched other cases during several months, and even years, without a fatal result supervening, and in such cases one might find obsolescent tubercle. He knew of one case which had apparently dated back thirty years.

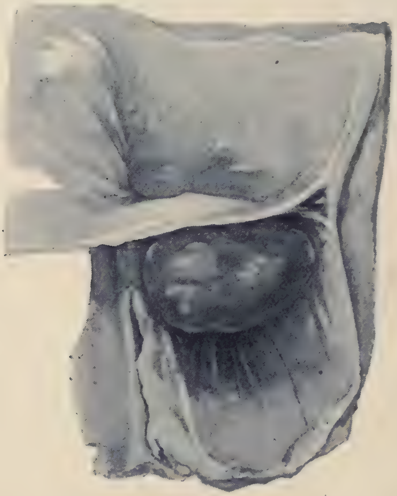
52. A TUBERCULOUS NODULE ARISING FROM THE DURA MATER.

By J. W. THOMSON WALKER, C.M.

The following is a short note on an unusual form of intra-cranial tuberculous nodule which was discovered *post mortem* in the case of Alfred C—, aged 2 years, at the Paddington-Green Children's Hospital.

Post-mortem examination.—The skull bones were thin and the inner table atrophied. Acute leptomeningitis was found over the convexity of both cerebral hemispheres and at the base of the brain. There was no distension of the cerebral ventricles. A tumour, the size and shape of a walnut, was found arising from the inner surface of the dura mater. The tumour, which was firmly attached to the dura mater by a constricted base, was of a yellowish-brown colour, and had a smooth nodular surface. It was situated in close proximity to the left lateral sinus, the torcular Herophili, and the occipital and straight sinuses, but there was no evident obstruction of these channels (*vide* Fig. 19). When the brain was in position the tumour was buried in the substance of the upper part of the left lobe of the cerebellum. On microscopical examination the tumour was found to be a caseous tuberculous nodule. A culture on serum-agar taken from the subarachnoid fluid at the base of the brain gave a growth of pure staphylococcus.

Fig. 19.



Tuberculous nodules are frequently enough met with in the substance of the brain, and are often multiple, but they rarely take the form of a tumour of the dura mater such as that just described. The final stage of these intra-cranial tuberculous nodules is usually an acute tuberculous meningitis; but in this case the distribution of the meningitis together with the absence of distension of the ventricles, the negative result of an examination for the tubercle bacillus in sections of the inflamed membranes and in cultures, and lastly the growth of a pure culture of *Staphylococcus aureus,* all point to a staphylococcal rather than a tuberculous infection of the meninges. The tumour had apparently produced little if any change in the intracranial pressure, for the symptoms were those of the acute meningitis, and no suspicion of the presence of the growth existed during life. The question of operation did not therefore arise, but had localising symptoms been present the nodule would have been in an exceptionally favourable position for removal, although the operation must have been complicated by the close proximity of the large venous channels. (*Exhibited April the 18th,* 1902.)

53. A CASE OF PRIMARY OSTEOMA OF THE BRAIN.

By J. W. THOMSON WALKER, C.M.

Rose F—, aged 9½ years, was admitted to the Children's Hospital, Paddington Green, under the care of Dr. Leonard Guthrie, on November the 9th, 1901.

History.—The patient was quite well till July, 1901, when she complained of severe frontal and occipital headache. There was considerable retching, but no vomiting. She remained in bed for three days, but was then able to return to school. The headaches, however, never entirely disappeared, and on October the 26th they became very acute in the frontal region, and three days later over the occiput. This time there was vomiting as well as retching, and the symptoms increased in severity until her admission. The family history had no bearing on the case.

On admission she was poorly nourished, though full-grown. She was dull and drowsy, but quite conscious and intelligent. Her expression indicated severe suffering. She lay with her head turned to the right, and resisted with a scream of pain any attempt to move it in the opposite direction. The pain seemed localised to the right occipital region and the back of the neck, and there was much tenderness on pressure over these areas. The pupils were equal but dilated and sluggish. There was intense double optic neuritis of long standing. There was slight ptosis of the right eyelid, but no other paralysis, muscular power being good and equal on the two sides.

The knee-jerks were absent, the plantar reflexes active and flexor in type. The urine, heart, and lungs were normal.

November the 12th.—Pain on the right of the occiput and in the neck is very severe, and ptosis is more marked. She complains of pain in the right arm, which is paretic and trembles on voluntary movement.

13th.—There is slight paresis of the right side of the face.

15th.—She is increasingly drowsy. Ptosis is very marked of the right eyelid. She sways to the right on standing with her eyes closed, and also on walking. The plantar reflexes and knee-jerks are absent. Pain and tenderness over the right side of the occiput is still most intense.

16th.—Mr. Murray trephined, to-day, over the seat of pain on the right side of the occiput. The meninges were very tense, but pulsation was present. The dura mater was not opened, but the removal of the bone gave immediate relief to her pain.

18th.—The dura mater was opened. A large quantity of clear fluid escaped. The cerebellum bulged, but looked quite healthy. Neither tumour nor abscess of the cerebellum was present.

The operations afforded prompt and permanent relief to pain, but no further improvement resulted. The child gradually became worse and more drowsy, and died somewhat suddenly on November the 28th. On the day preceding death there were profuse hæmatemesis and melæna. The temperature, which had been normal or subnormal throughout, rose to 104·2° F. two hours before death.

Remarks.—The symptoms were strongly suggestive of a lesion in the right side of the cerebellum. The cerebellum was, however, perfectly healthy, and the position of the tumour throws no light on the causation of the acute pain and tenderness in the occipital region.

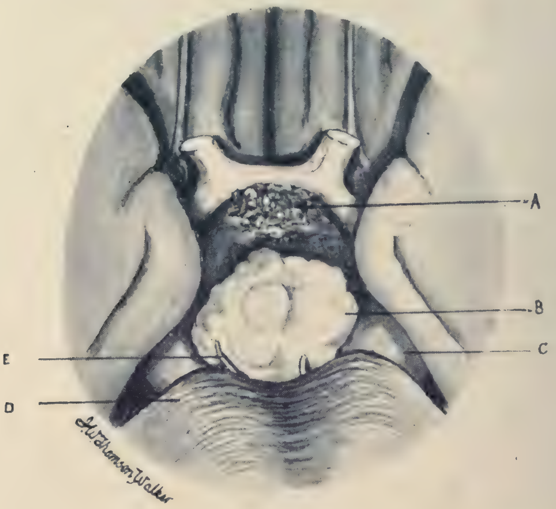
The bone, however, seemed somewhat thinner here than elsewhere, and possibly pressure from exudation of fluid was determined in this direction rather than in any other owing to adhesions which prevented equal distribution of the fluid in the cerebral cavities.

The case illustrated the value of palliative trephining, and the uselessness of localised pain and tenderness in the diagnosis of the site of an intra-cranial lesion.—LEONARD GUTHRIE.

Autopsy (November the 27th, 1901).—At the base of the brain the interpeduncular space is expanded, the crura cerebri being pushed backwards and the optic commissure forwards. The anterior third of the space presents an irregular prominence in the position of the infundibulum, the surface of which is irregular, and shows numerous projections of calcareous material (Fig. 20, A). Posterior to this prominence, and occupying about two-thirds of the space, is a cyst rather larger than a walnut (Fig. 20, B). The wall of the cyst is about the thickness of ordinary writing paper; in parts it is semi-transparent, and numerous particles and platelets of calcareous material adhere to its inner surface. The contents of the cyst were lost during the removal of the brain, but probably consisted of clear straw-coloured fluid as this was found in excess at the base of the skull after removal of the brain.

The cyst communicates, by an opening about the calibre of a goose-quill through the floor of the third ventricle, with a smaller cyst lying in the ventricle itself. This second cyst is about the size of a pea; the wall resembles that of the larger cyst, but contains no calcareous material. The communication between the two cysts passes through the floor of the interpeduncular space at the posterior perforated spot. The third pair of cranial nerves lie upon the under surface of the cyst (*vide* Fig. 20, E).

Fig. 20.



The irregular prominence at the anterior part of the interpeduncular space is found to be the under surface of a hard calcareous nodule about the size of a cherry. It corresponds in position to the infundibulum, and its upper surface forms a rounded prominence in the anterior part of the floor of the third ventricle. This upper surface is completely covered by brain tissue, and is smooth, unlike the irregular under surface on which numerous calcareous projections appear. The mass presses forwards on the optic commissure.

The third ventricle is larger than normal, the lateral ventricles are of natural size. The left lobe of the cerebellum is partly softened and disintegrated, the result of an exploratory operation. The brain as a whole appears unusually large for a child of this age. The base of the skull is normal, and there is no abnormality in the other parts of the body.

Microscopical examination.—The calcareous nodule was decalcified in von Ebner's solution, and sections cut in paraffin and stained with eosin and hæmalum, and with van Gieson's stain.

The mass was found to consist of spongy bone embedded in brain tissue without any well-marked capsule.

Three different stages are easily distinguished in the formation of the tumour, and the description is conveniently arranged according to these stages, beginning at the peripheral or growing part of the tumour and passing inwards to the fully formed bone.

1. *Cellular tissue.*—At the most peripheral part the microscopic field shows clumps of cells embedded here and there in brain tissue. The simplest of these cell groups consists of a small rounded mass of cells closely packed together. The cells have large, oval, deeply-staining nuclei and a relatively small amount of protoplasm, and are so densely packed that it is impossible to determine the presence of intercellular tissue. At the circumference the cells are set vertically, so that the long axis of the oval nucleus radiates from the centre. These peripheral cells are clearly defined from the surrounding brain tissue, and show no tendency to infiltrate it. The cells in the interior of the mass are irregularly and more loosely arranged, and their nuclei are large and oval and vary slightly in size.

In a second form of cell mass, which is apparently a further development of the first, the peripheral cells are definitely arranged in a single regular row; in the interior of the mass spaces lined with elongated nuclei are found, and are recognised as blood-vessels by the presence in them of red corpuscles. In some of these cell groups there is now an apparent tendency for the cells to become arranged in whorls, and in a few the inner cells have become star-shaped and resemble those of myxomatous tissue. Passing still further from the periphery of the tumour the cell groups become much larger, and in the centre of the whorls just described a substance, staining pink with eosin and having a finely granular appearance under high magnification, becomes apparent.

These nodules of finely granular material increase in number and size, and the cells which separate them become more and more flattened, and their nuclei elongated until at length only a trace of the nucleus may be found between the contiguous granular masses.

At this stage the cell masses are of comparatively large size, and have the following appearance. The outer layer of cells consists of a single row of vertically placed cells, with oval nuclei, closely packed together and clearly defined from the surrounding brain tissue. In parts this vertically placed layer of cells is pressed upon by the masses of granular material, and the cells have become flattened out. The mass is composed of cells with large oval nuclei surrounding nodules of granular material. This granular material, which is described in greater detail below, is in isolated nodules or in nodular masses. In some parts of the cell mass there are small areas of myxomatous tissue, but this is nowhere a striking feature; at other parts the cells are larger and somewhat irregular in shape. One or two large protoplasmic masses are seen with numerous closely packed nuclei, and these will be shown to be osteoclasts.

With the increase of the granular material the cellular elements become less and less prominent, until a stage is reached in which a round or laminated granular mass is found lying in the brain tissue and surrounded by a narrow ring of small, somewhat irregular, cell nuclei. In the brain tissue in which these cell masses are embedded there is an increase in the nuclei in the part immediately surrounding the mass, but no other change is observed.

2. *The granular substance.*—This material appears almost homogeneous under a low power, but under higher magnification its granular structure is apparent.

It is found either as round or oval nodules, isolated or closely packed together with a few nuclei between the nodules, or as irregular masses consisting of layers of short curvilinear bands with here and there a flattened nucleus interposed. Under high magnification this material has the appearance of large unstained spindle-cells, and with a more coarsely granular area representing a nucleus. In some parts of the sections the process of formation of this granular material may be traced. The cells become swollen, and appear as large oval or rounded protoplasmic masses which take the ordinary protoplasm stains poorly. The size of these cells greatly exceeds that of the ordinary cells of the cell mass, and the very faintly stained or quite unstained protoplasm contrasts strongly with that of the ordinary cells, but transition forms may be traced between the two. Granules now begin to appear at the periphery of the cells, and are apparently deposited in the protoplasm. This granular material increases in amount, and the cell form becomes less and less distinct until it is only recognised by a faintly indicated outline, and the nucleus by a more richly granular area in the centre of this.

The next step is the deposit of calcareous material in the granular masses, as indicated by the blue staining with hæmalum. This apparently begins at the periphery of the mass and spreads inwards. Ultimately the entire mass becomes calcified, and the nuclei are completely obscured by the blue staining.

Where the granular material is arranged in rounded masses these are separated here and there by capillary blood-vessels and a few cells, but no blood-vessels penetrate between the curvilinear bands.

At one part of the tumour a peculiar change has taken place, leading to the formation of a substance which at the first glance resembles the granular material. On close examination of this substance, however, it is found to be quite homogeneous, and there is no trace of the cellular structure found in the granular material. Cells lie embedded in this substance, either isolated or in groups, but there is no sign of transition from the cell to the homogeneous substance; it appears more like a secretion or an extensive hyaline degeneration than a part of the process described above. It is possible that the cavity containing this substance, the wall of which is lined partly by flattened-out cells of the cell mass and partly by nodules of the granular material, may be a small cyst, and that the homogeneous material may be the cyst contents changed during the process of hardening and fixing. This becomes the more likely since it is found that the wall of the large cyst described below has a very similar structure to that surrounding this homogeneous material.

3. *Osseous tissue.*—The first appearance of true bone formation is found as delicate strands of homogeneous substance between the layers of the granular material or at the circumference of the rounded nodules. These strands appear first at the peripheral part of the mass and pass inwards, they take a pink colour with eosin and a brilliant red with fuchsin. The granular material becomes surrounded and invaded by this substance and gradually disappears; nuclei are found in relation to these fine lamellæ, and become enclosed in spaces in the homogeneous material as bone corpuscles. Here and there a large mass of protoplasm with numerous nuclei, an osteoclast, is found lying at the edge of a trabecula thus formed. The osseous tissue is laminated and arranged in trabeculæ which enclose numerous round or oval spaces containing scattered rounded nuclei in a fine reticulum, and relatively large blood-vessels the walls of which consist of a single layer of endothelial cells. At the margins of the osseous tissue the contents of these Haversian canals are found to be continuous with the neuroglia in which the growing part of the tumour is embedded.

A microscopical examination of the wall of the cyst shows an outer layer consisting of numerous rounded and oval cells with abundant intercellular tissue containing fully formed blood-vessels, and an inner layer in which the first two stages above described may clearly be distinguished. Thus there is the same formation of granular material, in nodules and in laminated masses, and the deposit of calcareous material in this. The process stops short, however, of true bone formation, the only indication of the third stage being the appearance of the fine strands of homogeneous substance in and between the granular masses, which have already been recognised as osteogenic fibres.

The microscopical appearances presented by the tumour may be summed up as consisting of three stages, arranged in three fairly well-defined zones. The first stage is found at the peripheral zone, and consists of small masses of undifferentiated cells which enclose blood-vessels. In the second stage, the intermediate zone, there is the development of nodules of granular material in the cell masses, which increase in number and size and become calcified, and at the same time the cellular elements become reduced. The third stage consists in the appearance of fine osteogenic fibres in and around the calcified material, and the formation from those of the bone trabeculæ of which the central zone consists.

The different stages of bone formation are thus represented in the tumour, which may therefore be classed as an osseous tumour of the brain. The microscopical appearances do not appear to me to suggest that the tumour is malignant in nature. Embryonic cells are certainly present and constitute the groups forming the outer zone of the tumour, and if these were considered alone it is possible that they might be looked upon as sarcomatous in nature. When the whole tumour is considered, however, and the different stages followed to the regular formation of bone, it is apparent that these cell masses form merely the peripheral growing part of a simple bony tumour. There is no tendency to irregular growth and infiltration of the brain tissue, and the cellular masses apparently form the outlying part of the bony processes. There is nothing, in short, which would lead one to suppose that the tumour is an osteosarcoma, and, further, the proportion and character of the cellular elements point to it being an osteoma which is still in process of growth. The border-line between an osteoma which is still growing and an osteosarcoma, when considered solely from the microscopical appearances, is, I admit, ill-defined; but where the stages of bone formation are regularly observed, and the cellular elements are neither excessive nor irregular in their growth, the tumour may, I think, be looked upon as one of simple nature, provided no metastatic deposit is discovered or any primary focus found in some other part of the body.

A careful examination of the rest of the body in this case failed to reveal any other new growth, and I have therefore concluded that the tumour may be regarded as a primary osteoma.

It is well known that cysts are prone to occur in the neighbourhood of any form of tumour of the brain, and that the cyst wall may be composed of tumour tissue, or of apparently unaltered brain tissue with a thin lining membrane. The wall of the cyst in this case is clearly of a similar nature to the tissue of the tumour, although the process stops short of actual bone formation. It has been mentioned in describing the microscopy of the tumour that at one part there is an accumulation of a quantity of homogeneous material in the centre of one of the larger cell masses, and it has been surmised that this may represent a small cyst, the contents of which have been produced either by a secretion from the cells or by some form of degeneration. It seems possible that the origin of the large cyst may thus be explained. Be this as it may, it is evident from the structure of the cyst wall that it has arisen by some change in a part of the tumour.

There still remains a question as to whether the tumour originated in the brain tissue, or developed from the meninges or the skull bones and then became embedded in the floor of the third ventricle. The tumour was surrounded on all sides by brain tissue, except at its under surface, where a few calcareous spicules projected on the brain surface in the position of the infundibulum. These, however, showed no signs of fracture, and there was no adhesion of the dura mater at this part, nor did any abnormality exist in the bones at the base of the skull. I therefore infer that the growth took origin in the floor of the third ventricle, and was independent of the membranes and the skull bones.

Ætiology.—The origin of such tumours is obscure. Benjamin * states his belief that an osteoma observed by him arose by ossification of a lipoma, since it was surrounded by a layer of fat. He refers to this process as occurring in the lipomata of animals, but does not touch the question of the origin of the lipoma.

Virchow † considered that osseous tumours took origin in a chronic localised meningo-encephalitis. He apparently based his statement upon his observation, in many cases, of small sclerosed patches, and states that he has observed cases in which extensive bone formation has taken place in these sclerotic areas. He assigned no reason for the presence of such areas of chronic inflammation.

Bidder's ‡ hypothesis appears to me to be more acceptable. He believes the tumours are examples of the Cohnheim theory of the origin of growths from an aberrant nodule of embryonic tissue.

It is significant in the tumour described above that it lay in the position of the infundibulum, a spot in which, more than in any other part of the brain, such developmental errors might be expected to occur.

So far, then, as this tumour is concerned, I am inclined to take this view as being the most probable.

Osseous Tumours described in the Literature.

In the literature from the earliest times there are descriptions of "osseous tumours" of the brain, but very few of these can with justice be accepted as primary osteomata, and many of them cannot even be looked upon as true bony tumours.

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* Benjamin, 'Virchow's Archiv,' xiv, 1858, S. 552.

† Virchow, 'Geschwülste,' vol. ii, S. 96.

‡ Bidder, 'Virchow's Archiv,' lxxxviii, 1882, S. 91.

The sources of fallacy are numerous. The indefinite nomenclature and looseness of description of the earlier writers render their cases worthless when an attempt is made to collect examples of this form of growth.

It may seem superfluous to insist that no growth in which the tissue falls short of fully formed bone should be included under the category of osteomata, and that microscopical examination must be the final test by which the proof is established. Yet on examination of many of the reports on cases of "bony growths" and "osseous tumours" described, even at a comparatively recent date, it becomes evident that the presence of calcareous deposit has been deemed sufficient guarantee for the presence of bone, and that microscopical examination was either unobtainable or considered unnecessary.

The calcification of tuberculous nodules, of inflammatory exudations, of sarcomata, or of shrunken cysticercus cysts has, without doubt, led to many errors in this respect, and the observations of some of the older writers, such as Kentmann,[*] Shenk,[†] Kerkring,[‡] Meckel,[§] Greding,[||] and Walter,[¶] were undoubtedly of this nature.

The development of *true* osseous tissue in the brain substance may take place in a variety of ways other than primary osteomata, and such tumours must be carefully distinguished from the primary growths.

A certain number of cases have been described in which osteomata have arisen from the dura mater and become buried in the brain substance. Some of these tumours are small, but others have reached a very considerable size, and when large it is not always easy to trace them to their point of origin. They are most frequently found growing from the falx cerebri, and according to Virchow * more especially in the neighbourhood of the crista galli.

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[*] Kentmann, 'De calcul. in corp. human.,' obs. i, p. 1.

[†] Shenk, 'Obs. Med. Lib.,' i, obs. lxxvi, p. 113.

[‡] Kerkring, 'Specileg. Anat.,' obs. xxxv, p. 76, tab. xii.

[§] Meckel, 'Adami diss. et caussio quibusdam specialibus apoplexiæ.'

[||] Greding, 'Ludwigii adversar. med. pract.,' vol. ii, p. 475.

[¶] Walter, 'Obs. Anat.,' p. 42.

Hooper † illustrates very beautifully a number of these tumours growing in the falx cerebri, in the form of lamellæ and spicules of bone deposited in and projecting from this fold of dura mater.

Cases are also recorded in which these tumours develop on the inner surface of the dura mater over the convexity of the hemispheres. In the museum of Guy's Hospital there is a good example (No. 1475^{80}) of this form of growth. The note in the catalogue (vol. i) is as follows:

"Portion of brain with an irregular mass of bone about the size of a hazel-nut embedded near the surface. The membranes appear to have been thickened and adherent. It is situated at the top and near the middle of the head. It is not stated that it grew from the dura mater, but was no doubt connected with this membrane."

Numerous other examples of these tumours are given by Morgagni,‡ and the tumours collected by Voigtel § and Otto ‖ as osteomata of the brain also belong to this class.

Bony tumours growing from the skull bones and pressing into the brain substance have also been erroneously considered as cerebral osteomata. The tumours described by Sömmering ¶ and some by Otto ** were obviously of this nature.

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* Virchow, 'Geschwülste,' vol. ii.

† Hooper, 'Morbid Anatomy of the Human Brain,' 1828, pl. xii, fig. 7.

‡ Morgagni, 'De sedibus, etc.,' epist. xxv, art. 6. See also illustrations by Hooper, loc. cit., pl. v, figs. 1 and 3; and Baillie, 'Morbid Anatomy Engravings,' fasc. x, pl. iv, fig. 3.

§ 'Handb. d. path. Anat.,' 1804, i, 619.

‖ Otto, 'Lehrb. d. path. Anat.,' i, 1830, p. 238.

¶ Sömmering, Zusätze zu Baillie, 'Anat. des Krank. Baues,' S. 268.

** Otto, 'Seltene Beobachtungen,' H. 1, S. 79.

Again, bone may be formed as a part of a mixed tumour, such as that recorded by Oppenheim,* who describes a tumour the size of a fist situated in the right frontal lobe of a boy aged sixteen under the following name: Tumor chondro-ossificans — teleangiectodes — myxomatodes. In this tumour, however, it is not clear that the ossified portion was anything more than calcified cartilage.

Jacobsohn and Jamane † describe an osteosarcoma situated at the posterior two-thirds of the vermis and growing into the hemispheres of the cerebellum, but it is not apparent from the description what the origin of the tumour was, or whether it was primary or metastatic.

After a careful search through the literature I have been able to collect eleven additional examples of primary osteoma of the brain. Considerable care has been necessary in selecting the tumours, owing to the numerous fallacies of observation, of description, and of nomenclature referred to above.‡

The following synopsis may be given of the tumours.

Of eleven cases in which the sex is noted, seven were female and four were male. The ages varied from nine years up to seventy-nine years.

It seems to me not unlikely that, although only two of the cases were children and the others adults or aged persons, the tumours in most of these originated at an early age, and either remained stationary or grew very slowly in adult life. The following fact would certainly appear to give support to this view. In eight adult cases in which the clinical history is recorded, four had symptoms of nervous disease existing since childhood. Thus, adding the two cases of children, in six out of ten cases the tumour was either found in childhood or there were symptoms of a grave nervous lesion from an early age.

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* Oppenheim, 'Archiv f. Psychiatrie,' B. xxi, 1890, obs. xii.

† Jacobsohn and Jamane, 'Neurologisches Centralblatt,' 1895, p. 1153.

‡ Neumann's case quoted by Albers is apparently accepted by Ebstein as authentic. The description in Albers' key to his Atlas, on which Ebstein bases his opinion, is, however, too meagre to justify such reliance, and the tumour has therefore not been included here. For the same reason the tumour of Andral and Blandin (Andral, 'Path. Anat.,' ii, 849), which Ebstein considers doubtful, has been excluded.

The tumours varied from the size of a cherry-stone to a walnut, and were single ; and in this they show a marked contrast to osteomata of the dura mater and skull bones, which sometimes reach a very considerable size, and when small are very frequently multiple. In none of the cases was any cartilage present. The cerebral hemispheres were the seat of the tumour in five of the cases, the basal ganglia and floor of the third ventricle in three, and the cerebellum in four.

The following is a brief epitome of the cases.

1. *Benjamin* ('Virchow's Archiv,' xiv, 1858, p. 552).—Male, aged 32 years. Epileptic and imbecile. Bright's disease. Tumour occupied posterior part of corpus callosum, was covered by remains of corpus callosum, and extended into both hemispheres, pressed on right optic thalamus and right corpus striatum. The larger part was embedded in the right hemisphere ; the left cerebral hemisphere was markedly sclerosed. Surrounded by a well-marked layer of fatty tissue, and outside this a connective-tissue capsule. Microscopy.

2. *Bidder* ('Virchow's Archiv,' lxxxviii, p. 91).—Male, aged 59 years. Contracture of arm and leg since childhood. Tumour saddle-shaped, with irregular, nodular, prickly surface surrounded by connective tissue, and everywhere covered with brain substance. Lay in corpus striatum, and passed on each side into white substance of hemispheres with long axis (4 cm.) transverse. Brain otherwise normal. Full description of microscopy and illustration. Death from fracture of skull.

3. *Ebstein* ('Virchow's Archiv,' xlix, 1870, p. 145).—Female, aged 44 years. No previous nervous symptoms. Sudden cyanosis, paresis of right side, coma, and death in seven days. Tumour in substance of left hemisphere

of cerebellum, entirely surrounded by connective-tissue capsule except at one part where it lies directly under the pia mater. Surface nodular and prickly. Full microscopy.

4. *Meschede* ('Virchow's Archiv,' xxxv, 1866, p. 472; 'Zeitschrift f. Psychiatrie,' xxii, 1865, p. 415).—Male, aged 26 years. Epileptic and imbecile. Death from pneumonia. Tumour size of a walnut in anterior part of left frontal lobe, entirely embedded in brain tissue except at one point (three lines in extent) where it reaches the surface, and here soft meninges are lightly adherent. Surface rough, prickly. At one part a deep furrow filled with jelly-like myxomatous tissue. Some sclerosis of several parts of brain; number of thin cartilaginous-like plates in spinal arachnoid. In left hippocampus is a space the size of a half-bean communicating with lateral ventricle. Full description of microscopy.

5. *Ranvier* ('Bull. de la Soc. Anat.,' s. 2, vii, 1862, p. 219).—Spherical tumour, 2 cm. in circumference, completely embedded in the cerebellum between the superior and inferior vermis, surrounded by fibro-cellular capsule. Consists of two parts united by fibrous tissue; surface prickly. Microscopy.

6, 7. *Simms-Hooper* * (Hooper, 'Morbid Anatomy of the Human Brain,' 1828, pl. xii, fig. 7).—"A bony tubercle which consisted of the same materials as healthy bone with a little animal matter. There is a large internal cavity."

Hooper makes the following foot-note on the case:

"This was presented to the author by Dr. Simms, who removed it from an amaurotic girl ten years of age, who had been blind, and had tremors of her limbs for three or four years. It was found in the anterior lobe of the brain, surrounded by a purulent fluid, and is represented by me because it is an exact resemblance, only a little larger, of the one I found in the left lobe of an adult female, whose head I examined under circumstances which prevented me taking a drawing. It was the size and form of the eatable part of a walnut."

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* There is no microscopical description of these tumours, and the text is therefore quoted in full.

8. *Virchow* ('Geschwülste,' ii, p. 96).—Female, aged 27 years. Cherry-sized bone tumour in substance of left cerebral hemisphere, somewhat behind the middle, and not far from the surface. Surface nodular, mulberry-like, surrounded by firm fibrous capsule. Microscopy. No symptoms. Death from suppurative peritonitis in childbed.

9. *Virchow* (idem).—Female, aged 40 years. Paralysis agitans since tenth year on right side. Small, irregular nodule of bone situated in left optic thalamus.

10. *Virchow* (idem).—Old woman. Nine years' mental disease. Cherry-stone sized, yellow, ivory-hard tumour at anterior end of arbor vitæ of inferior vermis of cerebellum, immediately over roof of fourth ventricle; surrounding brain substance sclerosed. Microscopy.

11. *Witkowski* ('Archiv f. Psychiatrie,' xiv, p. 415).—Male, aged 79 years. No nervous symptoms. Bronchitis, bronchiectasis, nephritis senilis, gall-stones. Tumour walnut size, left cerebral hemisphere, shape of a double pyramid, surface grooved and prickly, surrounded by firm capsule, and entirely buried in the brain tissue. Surrounding sclerosis. External and internal hydrocephalus. Full microscopy.

(*Exhibited April the* 18*th*, 1902.)

Discussion.

Mr. Edmunds alluded to the fact that optic neuritis was said to be on one side only, and asked whether there was a similar condition in the other eye towards the end of the case.

Dr. Sutherland said the patient with the tuberculous nodule on the dura mater was in the hospital under his care ten days, and during that time the symptoms were those of tuberculous meningitis. He was surprised to hear that the autopsy revealed a tumour growing from the meninges, as he did not find any localising symptoms. There was well-marked papillitis on the right side, and such might be associated with a small tuberculous nodule which was sometimes the starting-point of tuberculous meningitis. In reply to Mr. Edmunds, he said only one examination was made, as the child was in a comatose condition. There was also weakness of the left side of the face a few days before death. Perhaps an opportunity of successful operation had been missed, but there were no localising symptoms of the tumour. He asked Mr. Walker whether he had any doubt in his mind as to the special form of meningitis which was present. He thought there was ground for strong presumption that there was a tuberculous basis in the case. There seemed to be another source of infection which Mr. Walker considered was staphylococcal.

Dr. Theodore Fisher (Bristol) thought the situation of the primary osteoma might have other interest. He understood that, apart from the cerebellar symptoms, there was no evidence of nervous trouble. It was thought disease of the pituitary body produced nervous trouble, and he had a case which seemed to bear out the theory. There was a gumma on the floor of the third ventricle which involved the whole of the pituitary body. The man gradually died in the course of six months without manifesting any definite symptoms beyond general weakness and mental lethargy. He had been thought during life to be suffering from granular kidney. At the *post-mortem* the only lesion found was a gumma of the floor of the third ventricle and the pituitary body. The kidneys were healthy.

Mr. Burgess (Harlesden) asked whether the urine of Mr. Walker's second case was examined, because he (Mr. Burgess) had at present under care a supposed tumour of the optic commissure pressing upon the third ventricle causing glycosuria and severe carbuncles of the right side of the face and right side of the chest.

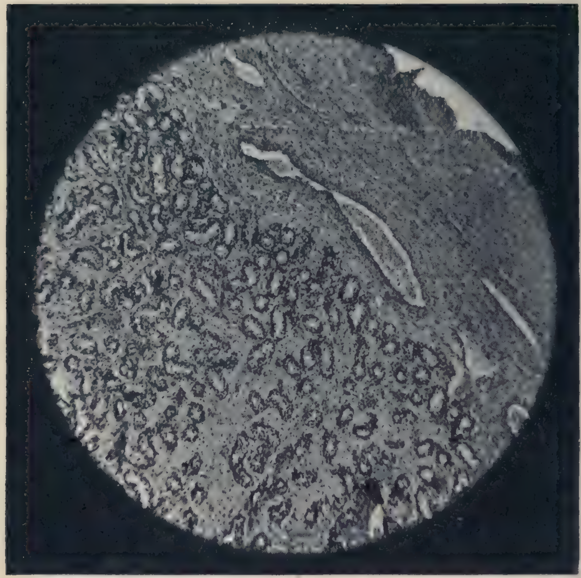
Mr. Thomson Walker, in reply, said he had felt doubtful as to the nature of the meningeal infection. All he had to go upon was, that a definite culture of staphylococcus was obtained, and he could not obtain any culture or other evidence of tubercle. Possibly there was a mixed infection, and the tubercle might have been missed. In his report he simply went on what was definite. In regard to the case of osteoma of the brain, he could not say much as to the influence of such tumours on the general health. Tumours of the pituitary body could produce symptoms like those of chronic Bright's disease, *i. e.* different stages of myxœdema. He understood the urine in the case he had narrated was normal.

54. NOTES ON A CASE OF DOUBLE INGUINAL HERNIA IN A FEMALE CHILD, EACH SAC CONTAINING AN OVARY-LIKE ORGAN.

By R. H. ANGLIN WHITELOCKE, C.M.

P. T—, aged 4 years, was seen in the out-patient department of the Radcliffe Infirmary, Oxford, on the 10th of July, 1900. Her mother stated that "the child had been very unwell for three days, had complained of pain in a swelling in her left groin, had been sick each day, and had refused to take any food." *On examination* there was seen to exist in the left inguinal canal a small oval swelling, tender to the touch, freely movable in the canal (which was extraordinarily long for a child of this age), but quite irreducible. The diagnosis of a strangulated hernia, probably of the left ovary, was made, and an immediate operation recommended. As the mother was very averse to any operation, I agreed to take her into the hospital and watch the case. After her admission the sickness seemed to abate a little, but the swelling remained irreducible, and became more painful. On the following day the mother gave her consent to the operation; the child was prepared, the usual incision for an inguinal herniotomy made, and the canal opened. A gland-like structure resembling a child's ovary, freely enveloped in a spacious sac, but quite irreducible through the internal abdominal ring, was found. It was attached by a distinct cord-like ligament, which could be pulled downwards through the internal ring for one and a half inches. There were no veins or other constituents representing a testicular cord, nor was there any other viscus discernible in the sac. It was not possible to ascertain whether the cord had been twisted upon itself, so as to give rise to the symptoms of strangulation; such a state of things has been described by Gifford Nash as occurring in a case of undescended testicle. The cord-like attachment was divided between two catgut ligatures and the organ removed. There did not seem to be any lumen in the section made. The sac was separated and pleated after Macewen's method, and a radical operation completed. Before the operation was finished the child began to cough, and this brought down a similar hernia on the other side, which was obviously freely reducible.

FIG. 21.



Seeing that no very apparent cause for the symptoms of strangulation had been found on the left side, I determined to operate on the right side at once. A similar operation (Macewen's radical cure) was performed on the right side, but in this case the ovary-like organ was returned to the abdomen, and not removed. The symptoms of strangulation at once ceased, and the child made a perfect and uneventful recovery. The wounds, which were not drained, were covered with a celloidin dressing, and healed by first intention.

Microscopical examinations of the gland prove that it is not an ovary, but an organ with a firm capsule made up of innumerable tubules embedded in a fibrous stroma (*vide* Fig. 21). The sections most resemble those made through an undescended immature testis.

This is, therefore, probably another of those cases of "hermaphroditism in congenital inguinal hernia," similar in many respects to those recorded by Chalmers, Martin, Neugebauer, and G. R. Turner. In external appearances this child is a girl. There are well-marked and normal external genitalia, and after making a rectal bimanual examination I am inclined to think there is an uterus present. In G. R. Turner's case there was atresia vaginæ and absence of the uterus. The organ removed, however, is certainly *not* an ovary, and histologically in most respects resembles an immature testis. I do not, however, think that there was a true vas deferens present, but I believe that it is a ductless gland. There was neither a Fallopian tube nor a plexus of veins or true spermatic cord on either side.

The points of chief interest are—

(1) The apparent hermaphroditism in a fine and otherwise healthy child.

(2) The presence of a double inguinal hernia in a female child so young, and in which the symptoms of strangulation existed just as if there had been a true enterocele. These symptoms were at once relieved by operative interference.

(3) As to the social aspect of this child's future: is she to be brought up as a male or a female? I hope to be able to keep her under my personal observation for some time longer, and watch her growth and development. The sections and photomicrographs shown were made for me by Dr. Arthur Waters, of Oxford, to whom I tender my sincere thanks.

It will be seen on examining the microphotographs, and sections under the microscope, that the organ is enclosed by a definite firm and vascular capsule; its structure is of a mass of tubules in a distinct stroma, presenting an appearance similar to what obtains in the testis of some of the lower animals.

(*Read April the* 18*th*, 1902.)

LITERATURE.

HOOPER MAY.—"Case of Hernia of Ovary in a Child seven months old," 'Brit. Med. Journ.,' i, 1898, p. 1389.

F. NEUGEBAUER.—"Quarante-quatre erreurs de sexe révélée par l'opération," 'Rev. de Gynécologie et de Chirurg. abdom.,' vol. iv, 1900, p. 459.

J. PINKERTON.—"Irreducible Hernia of Fallopian Tube and Ovary," 'Brit. Med. Journ.,' ii, 1899, p. 853.

BILTON POLLARD.—"Case of Strangulated Ovarian Hernia in an Infant aged three months," 'Lancet,' ii, 1899, p. 165.

G. R. TURNER.—"Case of Hermaphroditism," 'Lancet,' i, 1900, p. 1884.

Discussion.

Dr. George Carpenter asked whether Mr. Whitelocke made a rectal and bimanual examination of the child prior to the operation, because by so doing it was possible to make out the uterus, the Fallopian tubes, and the ovaries with the greatest ease.* Taking the utero-sacral ligament as a guide to the ovaries and Fallopian tubes, it would be possible to determine whether the ovaries were prolapsed into the hernial sac or not. He asked what was the condition of the uterus, Fallopian tubes, and ovaries. The ovary-looking organ exhibited was undoubtedly an infantile testis. It, however, contained much more stroma than was natural, and the tunica albuginea was very thick. It reminded him of a syphilitic testicle; it was like one he had illustrated in his book on 'Syphilis in Children,' and was very suggestive. He had examined several congenital syphilitic testicles, as also normal infantile testicles, and was struck with the resemblance to the former. Professor Cheine, of Edinburgh, had operated on a child with a swelling in the left inguinal region, and the contents of the sac were found to be an ovary and a Fallopian tube. If a rectal and bimanual examination had been made in that case, this, the youngest ovariotomy on record, might have been avoided.

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* "On the Value of Rectal Exploration as an Aid to Diagnosis," by George Carpenter, 'Pediatrics,' vol. i, pp. 481—507, June, 1896.

Mr. Thomson Walker said his opinion was that the doubtful specimen was a badly developed testis; the tubules were plainly testicular in arrangement, and it did not at all resemble an ovary.

Dr. Sutherland asked what Mr. Whitelocke proposed to do in regard to the child's future. That was an important matter to the child. As far as he could judge she should be brought up as a woman.

The Chairman (**Dr. Leonard Guthrie**) agreed with the view expressed by Dr. Sutherland.

Mr. Whitelocke (Oxford), in reply, said he did examine by the rectum, and investigated the case bimanually. He could not be quite sure he could feel the ovaries. He wished he had reached that stage of dexterity acquired by Dr. Carpenter in the matter, because he had encountered cases in which it was very difficult to feel the ovaries. He said he only met the case accidentally because it was thought to be one of strangulated hernia. He looked for a lumen in the cord-like attachment, but could find none, and there was an absence of the vas and pampiniform plexus. But there was at any rate an orifice, and there was also what appeared to be a hymen. In the few other cases on record there was a peculiarity about the urethral orifice, and the vagina had been a very short one. In Mr. Turner's case, which he had mentioned, the child was fifteen years of age, its vagina was only about two inches long, and there was no uterus. He intended keeping the child under observation, and making a further examination when she was older. The mother was a suspicious woman, and an attempt to push the inquiry would result in the defeat of his objects. He was glad to hear that the organ consisted of a tissue containing testicular tubules. If the tissue of an undescended human testis were put side by side with that of the lower animals, such as the pig, some expert histologists would be mistaken. He brought one of his collection of testes, from a patient aged fifteen, to view for purposes of comparison. Many surgeons held that undescended testes were very prone to become neoplastic. He had transplanted two or three such in recent years into the scrotum, and in two or three months they had developed and become very large, and felt very much like the normal organ. He thought those transplanted would one day become functional. As to how the child was to be brought up, Dr. Sutherland and the Chairman had ruled that she should be reared as a young woman.

55. A CASE OF TRANSPERITONEAL NEPHRECTOMY FOR SMALL ROUND-CELLED SARCOMA IN A BOY AGED FOUR YEARS.

By C. O. HAWTHORNE, M.D.,

AND

H. J. CURTIS, B.S.

The chief interest of this case is to be found in its surgical aspects. Though a favourable issue cannot be recorded, there is no reason to regret that an operation was undertaken. In it there resided obviously the sole chance of prolonging or saving the patient's life, and the fatal event arose from an unfortunate complication which, so far as can be judged, had no immediate or direct connection with the operation.

History of the case.—The patient, a boy aged 4 years, was brought to the Royal Hospital for Children and Women on October the 7th, 1901. He was then the subject of a considerable abdominal tumour, which, without much difficulty or hesitation, was recognised as a growth arising from the region of the left kidney. The enlargement of the abdomen, accompanied by undue prominence of the superficial veins, had only been recognised by the boy's mother as recently as September the 23rd, though it was allowed that the patient's health had not been satisfactory for a period of two months.

It appears that the boy accidentally fell when running in the street on August the 5th, and that subsequent to this he had never seemed quite well. This fall has the fortunate consequence of securing for us a knowledge of the boy's condition two months before he came under our own observation, as on August the 7th, that is two days after the accident, he was admitted to St. Thomas's Hospital. Through the courtesy of Mr. P. W. G. Sargent we learn that on that date the boy was reported to have been continually sick and fretful since his fall; that on examination he was noted to keep his legs drawn up on the abdomen; and that there was dulness on percussion with marked tenderness in the left lumbar region. In the course of the next day or two, however, the boy lost all his symptoms, and he was discharged apparently well on August the 10th.

From this date until the mother's discovery of the abdominal swelling in the latter part of September there appear to have been no symptoms of a definite character. The boy seemed weak and listless, and perhaps lost flesh, but he made no complaint of pain. At no time was blood observed in the urine. There was no albuminuria either when the urine was examined at St. Thomas's Hospital or by ourselves; and vomiting was limited to the two or three days immediately after the accident.

On admission he was a thin and delicate-looking lad of very quiet demeanour, and did not complain of pain or other discomfort. In the course of a few hours he seemed quite at ease, sat up in bed, played with toys, and was apparently entirely unaffected by the presence of the tumour. The family history presented nothing of interest.

Examination of the abdomen.—The superficial veins over the left flank were seen to be enlarged, but there was no œdema of the abdominal wall. A hard, irregularly rounded swelling could be felt almost half filling the abdomen, and occupying chiefly the left flank. Above the umbilicus it crossed the middle line and reached nearly to the right linea semilunaris. Below the umbilicus the tumour did not extend quite as far as the middle line. Its smooth rounded lower border dipped just below the iliac crest. No notch could be felt anywhere. The swelling did not move with respiration. Though generally of very firm consistence, in places it seemed soft and almost fluctuating. Its median half was resonant on percussion, the descending colon evidently lying over it.

The *urine* was of normal colour, of specific gravity 1026, of acid reaction, and free from albumen.

The *chest* was normal.

The *temperature* on admission was 96·6° F., rising three days later to normal.

Transperitoneal nephrectomy was performed on him four days after admission, the tumour, which involved the left kidney, being shelled out with a good deal of difficulty owing to the firm adhesions to the posterior abdominal wall (especially over the lower half of the back of the growth), and to the tail of the pancreas. There was in consequence considerable shock, but very little hæmorrhage. The corresponding ureter at its origin was completely concealed in the growth.

The tumour removed weighed 3 lbs. 15 oz. It was firmly encapsuled, and measured about six and a half inches from above downwards, and six inches from within outwards.

After recovering from the immediate collapse there was persistent vomiting, feeding having, in consequence, to be almost entirely rectal. Otherwise, during the first week the patient seemed to be doing well, the temperature remaining normal.

Five days after the operation the child began to suffer from cough. The temperature was 98·8° F., the pulse-rate was 136 to the minute, and the respirations 28 per minute. The chest was normal.

The wound closed, but owing to the persistent vomiting and the onset of cough the stitches were not removed before the eleventh day. The patient unfortunately developed pleurisy with effusion on both sides of the chest, and died on the fifteenth day after the operation.

The state of the urine was as follows:—On the same evening as the operation the amount of urine passed was 4 oz.; and the same amount was passed the next day, the specific gravity being 1030, the reaction acid, and there was no albumen. On the second day after the operation about 14 oz. were passed, but all of it could not be collected. On the next four days the amounts collected were 11 oz., 6 oz., 4 oz., and 13 oz. respectively. On the last of these days the specific gravity was 1028, the reaction was acid, and there was neither albumen nor sugar. On subsequent days the following amounts were collected:—5 oz., 4 oz., 10 oz., 8 oz., 8 oz., 7 oz., and 8 oz.; the bowels at the same time acted freely, once or more frequently each day.

At the autopsy the wound was found to have united throughout. There was no evidence of peritonitis. There were a few adhesions of the intestine to the parietal peritoneum where it had been divided in order to shell out the kidney tumour, the divided peritoneum having apparently readily united. So far as the nephrectomy was concerned the condition appeared perfectly satisfactory, the entire growth appearing to have been removed. There were no secondary growths or enlarged lymphatic glands. The right kidney and the liver were normal.

On opening the chest a considerable quantity of fluid was found on both sides, especially on the left side. There was extensive pleurisy of both pleural cavities.

Sections made through the lungs showed them to be practically normal.

There were no secondary growths in the brain or elsewhere.

Description of the tumour removed.—A median section carried through the growth and the kidney shows the tumour, as so frequently happens, to be arising from the connective tissue within the hilum of the kidney, the cortex of which, measuring nearly seven-eighths of an inch in depth, presents a fairly normal appearance. From the smooth rounded appearance of the tumour as it lies in contact with the cortex of the kidney, it seems evident that the renal pelvis, though distended with growth, is still intact.

A slit-like cavity in the region of one of the pyramids is suggestive of dilatation of one of the calices from back pressure.

The upper end of the corresponding ureter is embedded in the growth.

The tumour is limited by a double capsule, that of the kidney proper and that of the perinephric fat, the two layers being for the most part intimately blended, and of a dense white colour and very firm consistence. On the upper end of the tumour the normal supra-renal body, partly torn across in removal, is visible.

The cut surface of the growth is somewhat fibrous-looking, white, and homogeneous, but there are numerous areas in it of bright red blood extravasations. There does not appear to be any cystic degeneration.

When quite fresh the growth was very soft, granular, and extremely friable, readily escaping on slight pressure after incising the capsule.

Microscopically the appearance of the tumour is that of a small round-celled sarcoma. Rounded masses of sarcomatous tissue, containing many new vessels, lie embedded in a well-formed stroma of connective-tissue fibres, which only here and there show any tendency to develop into spindle-cells. A very few tubules can be seen surrounded by the round-celled sarcomatous elements. They are probably remains of glandular elements in the connective tissue of the hilum.

Remarks.—Such a soft, vascular, rapidly growing, round-celled sarcoma is the most common variety of all, and composed twenty-three out of forty-three tumours of the kidney operated on in children since 1890, according to Henry Morris.

As is well known, untreated malignant renal tumours are rapidly fatal in children, death following soon after the tumour has become obvious. As regards the diagnosis, it must be remembered that in children pain is often absent, and hæmaturia is also absent in three-fourths of the cases of malignant tumours of the kidney (Tuffier).*

Early diagnosis and an operation before the invasion of the tissues around, and especially of the lymphatics, are essential for the diminution both of operation mortality and of the liability to recurrence. Nephrectomy is useless in cases where the glands and other tissues around are infiltrated with secondary growth, and should this be found on making an exploratory laparotomy through one or other linea semilunaris the operation should be abandoned.

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* Quoted by Fenger in vol. ii of the 'International Text-book of Surgery,' by Gould and Warren, 1900.

It is noteworthy that though the first successful nephrectomy in a child was done only in 1877, by Jessop, and the operative mortality up to 1890 was as high as 60 per cent., since 1890 the mortality has been reduced by two-thirds, so that in 1898 it was only 17 per cent. (Henry Morris).

The advisability of surgical interference until recently was open to doubt; but two successful nephrectomies by Abbé, in 1892, and mentioned by Henry Morris,* are so interesting, and go so far to settle this vexed question, that we venture to quote them in full, though a considerable number of successful cases have also been recorded before and since the date above mentioned.

D. R. Abbé records† two cases of round-celled sarcoma of the kidney, one of them containing a good deal of involuntary muscle, which were cured by nephrectomy.

1. A girl, aged 2 years, was operated upon on April the 12th, 1892. The weight of the tumour was 2¼ lbs.

The patient was quite well at the age of five years.

2. A girl, aged 14 months, was operated upon on November the 20th, 1892. The weight of the tumour was 7½ lbs., or just one-half that of the weight of the child immediately after the operation.

She was alive and quite well in April, 1895.

One other feature of our case is worthy of note. It arises from the history of a fall some six or seven weeks before the detection of the tumour. Such a history, of course, raises the question whether the injury can reasonably be held in any way responsible for the development of the tumour. The apparent association between traumatism and the eruption of sarcomatous growths has often been observed. In a recent paper, Dr. Wm. B. Coley,* summarising his experiences, states that in his entire series of two hundred and seventy cases, eighty-six, or 31·8 per cent., gave a history of injury, and that in thirty-one, or 11·5 per cent., the tumour developed almost immediately after the injury. Whatever significance may be attributed to this association, the fact remains that in the present case the patient, within two months after a fall, at which time by a physical examination it was impossible to detect anything definitely abnormal, was then found to be the subject of a sarcomatous tumour arising from the left kidney, which nearly reached four pounds in weight. (*Read April the 18th,* 1902.)

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* 'Surgical Diseases of the Kidney and Ureter,' London, 1901.

† 'Annals of Surgery,' November 22nd, 1895.

Discussion.

The Chairman (**Dr. Leonard Guthrie**) said the authors were to be congratulated on the success of the operation, but were deserving of commiseration on account of the later development of pleurisy and death.

Dr. David Nabarro asked whether any infected glands were found in the abdomen at the *post-mortem*, and whether any striated muscular fibre was noticed.

Dr. J. P. Parkinson asked whether the left kidney was the one usually affected in such cases. Those he had seen were on the left side, but in some the right organ was enlarged also.

Dr. Theodore Fisher (Bristol) referred to a case in which both kidneys seemed to be affected simultaneously. Both kidneys were about double the normal size, and the trouble had apparently commenced in the hilum in each instance.

Mr. Thomson Walker said Mr. Curtis had apparently fallen into a usual mistake in joining together the adult and childhood cases in Tuffier's figures, whereas they should be treated separately. The prognosis of adult sarcoma of the kidney was very much the better. Some years ago, when looking through the statistics, he could only find two childhood cases, one of Abbé's and the other of Dr. Malcolm's, which had survived two years; but five or six cases in adults had survived as long as seven or eight years.

Mr. Burgess (Harlesden) asked whether Mr. Curtis could offer any explanation of the pleurisy with effusion.

Mr. H. J. Curtis, in reply, said he found neither infected glands nor trace of remaining disease at the *post-mortem* examination. He could not say whether the left kidney was more frequently affected ; he had only seen one other similar case, and in that also the left kidney was involved. He agreed that the statistics relating to children should be separated from those of adults. In the last edition of Mr. Henry Morris's work there were several more cases of prolongation of life beyond two years than Mr. Walker had mentioned. If that were admitted, it justified the operation of excising as soon as possible, because one never knew which case would survive beyond three years. It would not be good surgery to leave a renal sarcoma untouched. Laparotomy in certain cases had revealed apparently malignant tumours, and yet recovery, with complete disappearance of the tumour, had occurred, even in some few cases where portions of the tumours had been removed and were proved to be malignant.

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* Quoted in 'The Medical Review,' October, 1901, p. 597.

56. A CASE OF MALIGNANT ENDOCARDITIS, WITH HEMIPLEGIA AND EMBOLISM OF ONE ILIAC ARTERY, WHICH DEVELOPED CHANGES IN THE FUNDUS OCULI — OPTIC PAPILLITIS, A SIMULATED CHOROIDAL TUBERCLE, SUBRETINAL HÆMORRHAGES, AND PIGMENTARY DISTURBANCE.

By GEORGE CARPENTER, M.D.

ARTHUR E—, aged 7 years, had a strong family history of tuberculosis, and his brother, at the time of his illness, was in the Evelina Hospital suffering from tuberculous dermatitis, and died, whilst an inmate there, of morbus cordis.

The boy Arthur had been quite healthy, with the exception of an attack of measles, till July the 4th, 1901, when a rash was noticed "all over him," and he was feverish ; there was neither sore throat nor sickness. He was seen for the first time on July the 11th in the casualty department of the Evelina Hospital, on which occasion "his right leg was bent at the knee, and stiff, and the knee-jerk was exaggerated on this side." A systolic mitral bruit was heard, which was conducted into the axilla, and he was short of breath.

On July the 17th he was taken ill suddenly whilst at play. He came indoors to his mother, complaining of headache, and was put to bed. Almost immediately he had a "fit," and became unconscious. He twitched in the "fit" all down one side, but which was not certain, and he remained unconscious until he was admitted into the Evelina Hospital on the following day, when he was found to be twitching on the LEFT side.

On admission on the 18th he was comatose, his eyes were sunken, his lower jaw relaxed, and his left side paralysed, the lower half of the face being involved. The knee-jerk was absent on that side, but it was present on the right, the arm and leg of which he moved freely. There were no convulsive movements. The right pupil was widely dilated, and did not react to light; the left reacted, though badly. The cardiac impulse was in the sixth interspace, 1½ inches outside the nipple line, and unaccompanied by extension of dulness to the right; but there was some epigastric tenderness. A loud systolic mitral bruit was audible, which was conducted into the left axilla. The lungs were normal. The liver and spleen were not enlarged, and the urine was free from albumen. The pulse was slow, weak, and irregular; the breathing was also slow, and occasionally sighing. Sweating was profuse and general. The temperature by the rectum was subnormal. The abdomen was sunken (scaphoid), and tender to the touch. He was sick twice during the day.

July the 19th.—He was very sick during the early part of the night, after which his left arm was rigid for a time, and the other limbs were slightly so. He subsequently vomited half a dozen times. During the day "he moved all his limbs," and there was no rigidity. He was semi-conscious, and very irritable when disturbed. The pulse was very slow, but less irregular, and the pupils were not so unequal. A note on the 20th stated that during the night he moved the right side of his body more than the left, and his left leg was at times rigid. He continually drew his right hand across his forehead, and was still very irritable when disturbed. The pulse was slow and irregular; the pupils as before; and during the day the general condition was much about the same. His temperature, which had all the time been subnormal, rose to 100° F., and on the 21st it was 102·2° F. The fundus oculi was examined, as it was thought he was developing tuberculous meningitis, and marked optic papillitis was discovered. The sickness had ceased. He did not resent being disturbed. The arms and legs were rigid at times, the hands clenched, and the eyes fixed, and occasionally there were muscular twitchings. The pulse was rather quicker, but still weak and irregular, and he was more feeble. He had been fed by the nasal tube, which was still being used. On the 22nd he had been very quiet all night, his hands were clenched, and his limbs rigid, and he occasionally drew up his right leg. The abdomen was very scaphoid. During the day he scarcely moved at all, his hands were still clenched, and the limbs occasionally rigid. On the 23rd he had passed a peaceful night, and was quiet all day, occasionally moving his right arm over his head. On the 24th he had been very quiet all night, and slept with his eyes half closed, until the early morning, when he commenced to groan and scream, and drew his right hand across his forehead—this lasted for about an hour. He now took a little food by the mouth, and appeared to understand when spoken to, but he was unable to speak. He could not open his right eye (ptosis), but the left was constantly open. The left side of the body was rigid, and he moved his right leg and arm quite easily. The pulse was still weak and irregular. His temperature had been gradually falling, and on this day was once more subnormal. On the 25th, during the early part of the day, he was quietly sleeping, but in the evening he cried out and drew up his right leg, and put his right hand to the right side of the head, as if in pain. He was unable to move the left side. On the 26th he occasionally cried out and drew up his right leg. He could open his right eye a little wider. He constantly rubbed the right side of his head. He was fed partly by the nasal tube and partly by the mouth. His general condition was much about the same. On August the 1st he was quite conscious all day, and talked incessantly. He frequently waved his right leg, and his right eye was more open. He moved his left arm a very little, and the knee-jerk on the left side had returned, and was increased. On August the 6th the optic discs were swollen, about three dioptres, and the vessels were in places partly buried in exudation. A little more than the swollen diameter of the optic disc distance in the left eye, in the upper and inner quadrant, was a small pear-shaped patch of a light fawn-colour. It had to be carefully looked for by the direct method, as the difference in colour tone between it and the red reflex was not so marked as to make it appear a *very* staring feature, though quite striking enough; but by the indirect method it stood out well, and readily attracted my attention. I considered it to be a tubercle in the choroid, though, in my experience, its shape was not quite classical, and perhaps it was not quite obtrusive enough; but, apart from these criticisms, it was very like indeed, and I was quite prepared to accept it as a tubercle, and did so, founding a diagnosis of tuberculous meningitis on my discovery.* The optic papillitis was the pronounced variety usually associated with cerebral tumours, and I thought it possible he had a cerebral tuberculous tumour and secondary tuberculous meningitis. He could not now move his right eye in any direction, and he could only look towards his nose with his left eye.

August the 12th.—Since August the 1st he has had a good appetite, and has slept satisfactorily. He is beginning to move his eyes a little more now, but both external recti are paralysed. There is loss of tactile sensation below the left knee, and diminished sensation over the whole

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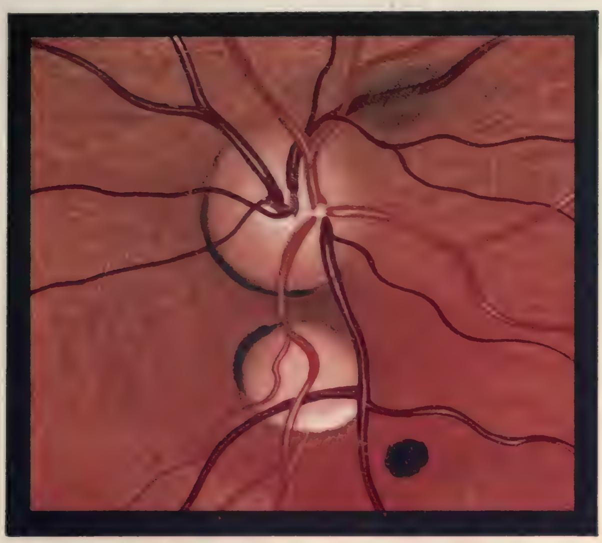
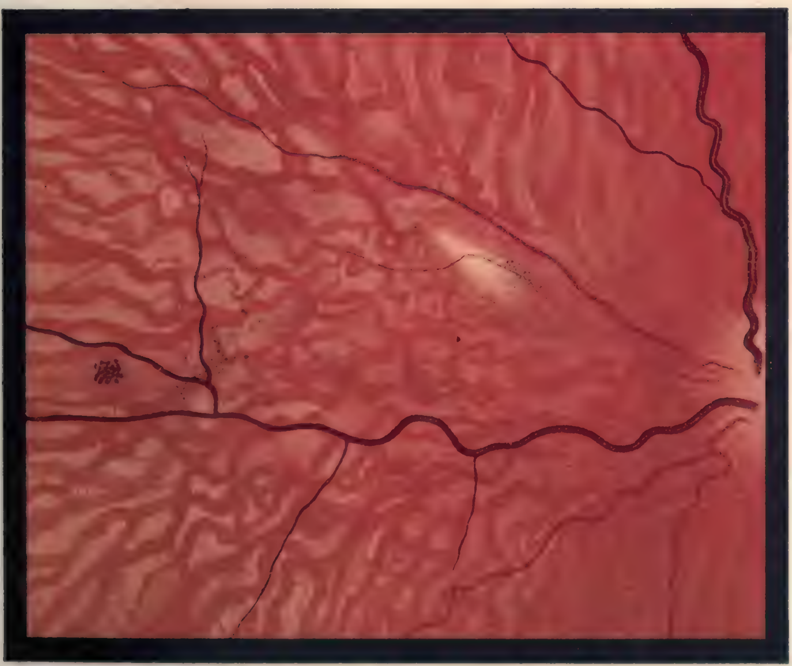
* *Vide* Plate VI. Compare this figure with figs. 3 and 5, plates viii and ix, between pages 172 & 173 and 174 & 175, vol. i, 'Reports of The Society for the Study of Disease in Children,' article "Tuberculosis of the Choroid," by George Carpenter, M.D., and Sydney Stephenson, C.M.



Mr. Sydney Stephenson's case of Tubercle of the Choroid; right eye, Alice P., aged 8 years.



Dr. George Carpenter's case of Malignant Endocarditis showing a simulated Tubercle of the Choroid, a subretinal hæmorrhage, pigmentary disturbance and optic papillitis; left eye, Arthur E., aged 7 years.

Bale & Danielsson, Ltd., Chrom. Lith.

of the left arm. The temperature up to this date has been either normal or subnormal; there have been one or two trifling rises, but they have not exceeded 100° F.

August the 15th.—Movements of the left arm and leg have greatly improved. In the former there is an all-round improvement, and in the latter they are best at the hip-joint. The knee-jerk is exaggerated, but knee-clonus cannot be obtained; there is, however, ankle-clonus. Sensation to pain is normal. Tactile sensation is much better in the leg. In the arm tactile sensation and sensation to pain have improved. The lungs, spleen, and kidneys are normal. There is now paresis of the right external rectus, and a linear hæmorrhage on the outer side of the right optic disc. Towards the periphery of the inner hemisphere of the left fundus there is some faint diffuse pigment.

August the 29th.—The boy is still rather sleepy-looking, and he is pale. The left side is weaker than the right, but there has been marked improvement in the paralysis since massage was commenced on the 9th of August last. He has recovered from the facial paralysis, and the ocular movements have become natural. The knee-jerks on both sides are brisk, being most marked on the left. Sensation is now normal.

September the 2nd.—The tubercle-like body in the left fundus oculi is more defined than has previously been the case. In the inner hemisphere, in the periphery, is a small bunch-of-grapes-like hæmorrhage, in which two minute retinal vessels terminate. Above this, and to its central side, round pigment-granules are freely sprinkled over a limited area.

September the 6th.—It was decided to inject the boy with tuberculin. Subsequent to August the 12th his temperature began to rise little by little, the highest point reached being 102·4° F.; it was intermittent. In the morning it was subnormal, usually somewhere between 97° F. and 98° F., and in the evening mostly between 100° F. and 101° F.; sometimes more and sometimes less. The pulse during the subnormal stages varied between 96 and 112 beats to the minute, and during the rises of temperature between 112 and 160 beats to the minute. In the apyrexial periods the respirations varied between 20 and 36 times to the minute, and during the pyrexial between 32 and 56 to the minute. Tuberculin (·003 c.c., supplied by the British Institute of Preventive Medicine) was injected at 2 p.m., when his temperature was 98·6° F.; at 4 p.m. it had reached 101·4° F., and by 6 p.m. was 101·6° F., and at 12 p.m. 99·8° F. Apart from the rise of temperature, which was but slightly in excess of what had already happened, nothing occurred. The fundus oculi was unaffected by it. A trace of albumen, unconnected with the injection, was discovered in his urine to-day; it had been tested on many occasions previously, and had always been found natural.

September the 8th.—He was very drowsy.

September the 9th.—He complained of pain in the right side of the abdomen at night, and was drowsy during the day.

September the 11th.—He was very drowsy all day. Erythematous patches appeared over the body, limbs, and face, and his eyes were bloodshot.

September the 12th.—The erythematous rash was fainter on the face, but there was more of it on the body.

September the 16th.—To-day there are a number of minute pigment-granules dotted over the choroidal "tubercle," and its condition has therefore changed in the interim, and in this respect it has conformed to the custom of choroidal tubercles. On comparing the grasp of the left hand with that of the right, there is still some weakness there. The knee-jerk on the left side is brisk, and there is ankle-clonus; the same applies to the right leg. He cannot read, but he can see pictures, and is interested in them. He is still very drowsy.

September the 19th.—Several examinations of the urine have been made since the 9th inst., and the specimens have been found to be free from albumen. To-day he is very drowsy and fretful, and his face is pale. Erythematous spots are to be seen on the feet, legs, trunk, and face. The left leg shakes when it is touched.

September the 20th.—The erythema is still present.

September the 21st.—He complains of pain in the right knee when it is touched, and he is very drowsy.

September the 23rd.—Erythematous blotches are again appearing on the face and body.

September the 24th.—The erythema is the same; his face is pale.

September the 25th.—There are fresh crops of erythema.

September the 26th.—He complained of pain in the right hand during the night, and now complains of pain in the right big toe. The face and legs are covered with an erythematous eruption.

September the 28th.—The rash is the same. He complains of pain in the right little toe.

September the 30th.—The boy is brighter, and he has a good appetite. He complains of pain in the lower part of his back.

October the 5th.—He has been sleeping quietly, and has relished his food; but is very fretful when he is disturbed.

October the 7th.—To-day there is a slight trace of albumen in the urine; he is very drowsy.

October the 10th.—He is very fretful to-day, and his extremities, at times, are cold. Erythematous blotches are reappearing on the face and legs. The pulse is weak.

October the 11th.—He slept badly, and was very sick once during the night and once during the day. He is very fretful, and is crying with pain in the left side of the abdomen. His face is alternately pale and flushed.

October the 12th.—He had a bad night, and suffered with great pain in the left side, which left him during the day, and he took his food fairly well.

October the 13th.—Another bad night, and crying with pain in his head. Retching. Stiffness of the limbs was more noticeable. He was sick once during the night, and sick again during the day. The child is drowsy, fretful when awake, and irritable when disturbed. The pulse is weak.

October the 15th.—The urine was of specific gravity 1024, and there was a slight trace of albumen in it. He was very bright during the day.

October the 20th.—For the past three weeks the temperature has been oscillating, usually remittent, and latterly sometimes intermittent. It has but once fallen below the normal during the first two weeks. For the first week it mostly varied between 101° F. and 103° F. for the highest rises of temperature, and for the remaining two weeks between 101° F. and 102° F. For the last week it has often fallen to subnormal, and has but once exceeded 101° F.

October the 22nd.—He was moaning and coughing frequently during the night. To-day he complained of pains in different parts of his body, but the resident-medical-officer did not find anything to account for them. He moaned continually, his pulse was rapid, and his breathing laboured.

October the 28th.—The urine was of specific gravity 1028, and free from albumen.

November the 1st.—He has been better recently and is taking his food fairly well. His nutrition is poor, he is very pale, and his finger-nails are blue. Both knee-jerks are brisk, and ankle-clonus can be obtained on both sides. He is occasionally crying and moaning, and complains of pain in his head and face.

November the 2nd.—He is drowsy and fretful.

November the 4th.—He is very languid and drowsy.

November the 6th.—The boy is drowsy. His urine is of specific gravity 1024, and free from albumen.

November the 13th.—He is fretful, and was groaning in his sleep nearly all night. His face is very pale. The urine is of specific gravity 1018, and free from albumen.

November the 15th.—He has taken his food fairly well, but is drowsy.

November the 16th.—He slept well, but was sick after one meal.

November the 18th to the 26th.—He was very sick once during the night of the 18th. Between these dates slight traces of albumen were found in his urine. He was sleeping well, and taking his food satisfactorily, but was occasionally sick. On the 22nd his eyes were examined, and his optic discs were found to be turning white. There was no obvious squint or defect in the extrinsic muscles; the pupils were semi-dilated, and almost without direct action to light. For a month or more the temperature has been oscillating, and has been more often remittent than intermittent; it has only twice been over 102° F., and on many occasions it has been subnormal.

November the 27th.—He was restless and fretful during the night, and groaned continually. To-day he is a little brighter, but his right hand is swollen and blue.

November the 28th.—He did not get to sleep till four o'clock this morning; his cough was very severe during the night, and there was much expectoration. His hand is much better to-day.

December the 1st.—He slept well. All the morning he was very drowsy, and took his dinner with persuasion. At 1.20 p.m. his left arm and leg became convulsed, and he became unconscious. The pupils were unequal, the right being larger than the left. This condition continued until 2.45 p.m., when he was sick, since when he gradually returned to consciousness, but he was unable to speak. He was very restless, and constantly sat up in his cot, and cried out as if in pain. At 5.30 p.m. he asked for drink, and his speech gradually became quite distinct. He then complained of much pain in his head and abdomen.

December the 2nd.—He slept fairly well, but was fretful and restless. In the small hours there was slight twitching in the left foot. All day he was drowsy and difficult to rouse. He complained of headache, and there was slight facial paralysis (incomplete) on the *right side?* The left arm was decidedly weaker than the right. The knee-jerks and clonuses were as before, and the legs were painful. Speech was very tremulous, slow, and almost unintelligible at times. The lips were blanched.

December the 3rd.—He passed a very bad night—cold sweats, and continually calling out. The urine was free from albumen. To-day he is restless and fretful, and moaning continuously. The left foot is œdematous. The right foot and leg are covered with blue patches, somewhat similar to a measly eruption. The toes are cyanosed and cold. He has been sick once.

December the 4th.—He was constantly calling out during the night, and very restless. All day he has been sleeping. The eyes are puffy.

December the 6th.—The left foot is less œdematous than it was. The pulse is occasionally barely perceptible at the wrist. He now and then complains of pain in the head and legs, and he lies curled up, turning first on one side and then on the other.

December the 8th.—He was sick once during the night, but slept well. He complains of feeling tired. His urine is free from albumen.

December the 9th.—His face is puffy, and he has a troublesome cough. The blue patches are more pronounced on the right foot, and the toes of the left are cyanosed. He is very drowsy and irritable, and his speech is very slow and incoherent.

December the 10th.—He slept well. His face is more puffy, and his cough is very troublesome.

December the 11th.—He slept well during the night; but from 7.30 a.m. he was continually turning from side to side, or sitting up in his cot. His breathing was laboured, and his alæ nasi dilated. The face and lips were cyanosed, and many coarse crepitations were audible over the bases of both lungs. The pulse was barely perceptible, and his extremities were cold. He was continually muttering and screaming, and gradually his speech became quite incoherent. His pulse was then imperceptible, and at 1.55 p.m. he was slightly convulsed, and died five minutes later, having been in hospital for 145 days. There was but little, if any, alteration in the character of the fever until within a week of his death, when 99·8° F. was the highest point registered, and the temperature was for the most part subnormal.

Post-mortem examination.—Brain.—The pia mater was much injected with blood-stained effusion over the whole surface of the brain. The convolutions were flattened, and much blood-stained fluid escaped on removing the brain from the skull. The base of the brain was covered with a hæmorrhagic gelatinous exudate, as was the vertex. There was no thickening of the pia mater, or any sign of tubercles along the vessels. The posterior part of the right cerebral hemisphere from the fissure of Rolando almost to the tip of the occipital lobe was considerably depressed. On opening the brain this depression was seen to be due to a "cyst" about the size of a walnut which was present in this region, extending forwards to about the middle of the lateral ventricle, and backwards to the limits of the posterior cornu of the ventricle. Greenish fluid with broken-down brain substance was present in this "cyst," which was separated from the posterior cornu of the ventricle by a thin partition of white matter. The ventricle itself was somewhat dilated, but otherwise looked healthy. Behind this cystic area the white matter was more solid, but dark reddish-grey and dirty-looking—evidently necrotic. A great part of the white matter of the right occipital lobe was involved in this necrosis, as was also a small area of the grey matter of the posterior parietal convolution (*vide* p. 214, Fig. 22). The middle cerebral artery was carefully examined for an embolus, but it could not be found. Presumably the necrosis and softening which ensued had involved the region of the embolism, and so obliterated any trace of it. The rest of the brain was normal.

Respiratory system.—The respiratory system was practically normal, except for a doubtful infarct in the upper lobe of the left lung. No emboli could be found in the pulmonary arteries.

Circulatory system.—The heart was considerably enlarged, and a few petechiæ were present on its surface.

The right auricle was healthy; the tricuspid orifice dilated. The right ventricle was hypertrophied, and the valves healthy. The left auricle was dilated, and its wall hypertrophied. On looking into it, the mitral orifice was found narrowed so as to admit a cedar pencil, the narrowing being due to the presence of a mass of "warty" vegetations projecting into the cavity of the auricle from the mitral valves. The left ventricle was much dilated; its wall was not much hypertrophied, and it was thinned near the apex. Projecting down from the mitral orifice into the ventricle was a large mass of vegetations, many of them a quarter to half an inch or more in length, and ribbon-like,

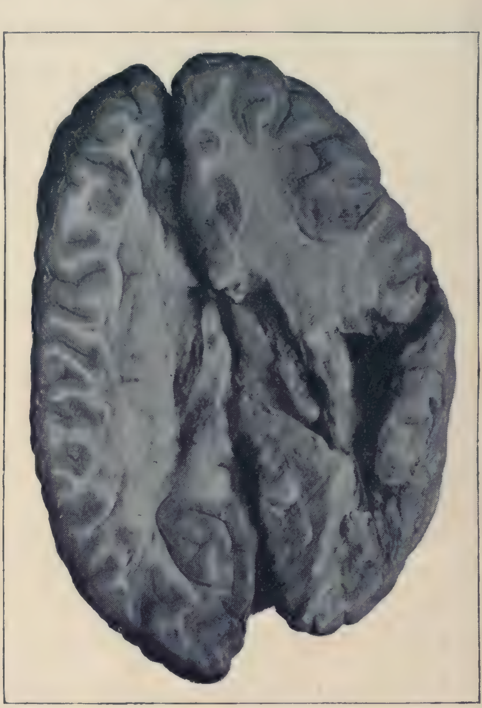


FIG. 22.—View of brain, showing large area of softening and destruction of brain substance.

being attached at one end, and otherwise hanging free in the cavity. On cutting open the mitral orifice, the opposed and auricular surface of the cusps was seen to be covered with sessile and pedunculated vegetations (*vide* Fig. 23). Large numbers of streptococci were seen in stained sections of these vegetations. The rest of the ventricle and the aortic valves were normal. The aorta, the left common iliac, and the external and internal iliac arteries were healthy. At the bifurcation of the right common iliac artery was a firm embolus which had eroded, and was closely adherent to, the walls of the artery. Spreading up from this embolus was a firm, decolourised, *ante-mortem* clot, more than an inch long, occupying nearly

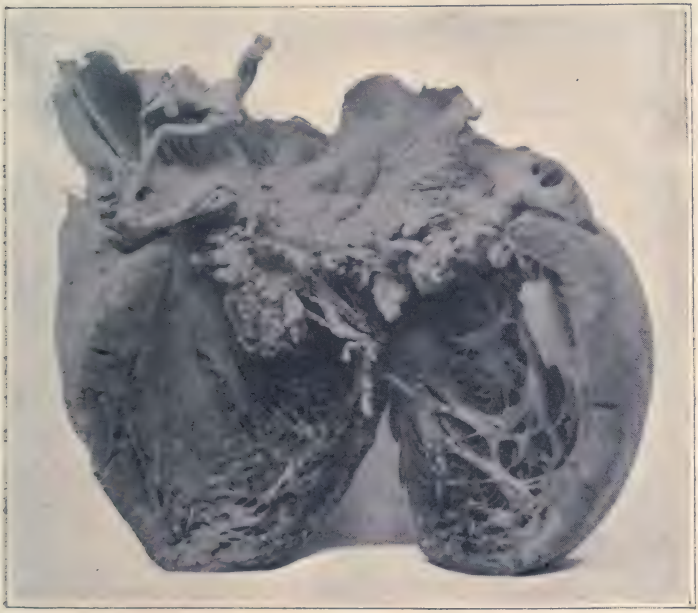


Fig. 23.—View of left auricle and ventricle. The mitral valve is extensively diseased, and covered with warty vegetations. The left ventricle is considerably dilated and somewhat hypertrophied. Some small vegetations are present on the auricular surface.

the whole length of the right common iliac artery. A small red clot (possibly *post-mortem*) extended down into the right external iliac artery from the embolus at the bifurcation of the common iliac. The internal iliac artery on the right side was occupied by a clot about one inch long, and decolourised; it was breaking down and almost purulent. A piece of the embolus at the bifurcation was stained for organisms, and showed streptococci. The adjacent right common iliac vein contained a long dark clot (*post-mortem* clot), and its walls were apparently healthy.

Alimentary system.—The œsophagus, stomach, and intestines were normal, except for many small, circular, red patches in the small intestine; these were either small submucous hæmorrhages or hæmorrhagic solitary follicles. The spleen and kidneys showed many infarcts, recent and old. The liver was large, somewhat pale and soft, but otherwise normal. The other organs were apparently healthy.

Microscopical sections of the eyes were prepared by Mr. Sydney Stephenson, and it was found that the fawn-coloured patch in the choroid was not a tubercle, and that there were subretinal hæmorrhages, proliferation of the retinal pigment, and considerable optic papillitis, as detected during life with the ophthalmoscope.

57. A CASE OF PAROXYSMAL HÆMOGLOBINURIA.

By JAMES TAYLOR, M.D.

The patient, a girl of 8 years, suffered from attacks of shivering and pyrexia, and at these times it was noticed that her urine was very dark in colour—indeed, it was described as being black. There was also cyanosis, and sometimes pallor of the extremities. She had several such attacks while in the hospital, but remained free from them if she was kept in bed. The urine was very dark, almost porter-coloured, and the deposit showed the presence of oxalates and the débris of blood, but no corpuscles. Oxalates were also present in the intervals between the attacks. There was no sign of congenital syphilis about her until a year after she came under observation, when she had syphilitic ulceration of the palate, which has resulted in destruction of the uvula.

(*Exhibited May the* 16*th*, 1902.)

58. A CASE OF RAYNAUD'S DISEASE.

By JAMES TAYLOR, M.D.

The patient is a boy of 5 years, whose fingers were noticed to be blue and cold at times since he was two years old. They also ulcerated, and so did his ears. He has lost the terminal phalanges of several fingers. His toes have ulcerated, but there is no loss of substance. He has no hæmoglobinuria, but oxalates are present in his urine in abundance. He has a soft systolic murmur at the cardiac apex. His mother died of "ulceration and mortification of the legs" at thirty-four years of age. A sister suffers from Raynaud's disease in the fingers only. (*Exhibited May the* 16*th*, 1902.)

Discussion.

Dr. James Taylor, in reply to the Chairman, said some of the attacks in the case of paroxysmal hæmoglobinuria were associated with blueness of the fingers, and the aunt stated that during the attacks the fingers were "like dead fingers." The family history of that case was curious. The father was dead and the mother was alive. The mother had first of all two miscarriages; then a child born at full term, but it died almost immediately; then another was born at full term, but died at the end of five months. The present was the fifth and only living child.

59. A CASE OF SPORADIC CRETINISM SHOWING IMPROVEMENT UNDER THYROID TREATMENT.

By HERBERT CARRE-SMITH.

G. M—, aged 12 years. This patient is from Dr. George Carpenter's clinic at the Evelina Hospital, and it is by his courtesy that I show her. This case has been under observation for four years. When first seen in March, 1898, she had the typical signs of myxœdema. She was originally brought for treatment on account of her excessive size, helplessness, and stupidity. She had always been dull and stupid, but only since Christmas had the myxœdematous signs and symptoms been so well marked. The head was flattened and broad from side to side, the hair being scanty and coarse. The eyes were wide apart, being deeply sunk in the surrounding œdematous tissue. The nose was flattened and broad at the base, the lower jaw massive, the lips thick, a short neck, large ears, and a muddy complexion; the tongue was large, and almost filled the mouth. The face was generally swollen, all parts being equally affected, the œdema being resilient and not pitting on pressure. The limbs were cold and reddish-blue in appearance; both legs and arms were mottled, and there were some blisters on the dorsum of the right foot. The subcutaneous tissue all over the body was much swollen, and the hands presented the typical spade-like appearance. Well-marked fatty pads were felt in the supra-clavicular regions; the glands below the jaw were enlarged; the thyroid gland could just be felt; the abdomen was pendulous. The patient was difficult to rouse, taking little interest in anything, sitting all day long staring into vacancy, and was irritable when disturbed. She understood simple questions, but took a long time to answer them, and then in a very slow, monotonous manner.

Past history.—She did not walk until she was six years old, and did not talk until five and a half years old. The teeth did not appear until the sixth year. She had always been delicate and deficient in mental power, and backward generally; she had attended school at intervals for about twelve months, and during that period had not finished learning her A B C.

Family history, etc.—The father and mother are both healthy, and have always lived in London; there are ten other children alive, and they are all bright and healthy. Two others died in infancy, one at six weeks of bronchitis, and one at seven months of asthma; the mother has had one miscarriage, last December.

Treatment.—The patient was placed under treatment on March 8th, 1898, and was given one and a half grains of thyroid extract in the form of tablets three times a day. She improved under treatment and the weight decreased; for instance, in one series of observations she decreased 10 lbs. in thirteen days, taking full diet.

Unfortunately the patient has not had systematic treatment with the thyroid-extract. A great difference has always been noticed when the treatment has been discontinued, the patient becoming "lumpy and mopy," as the mother describes it; and there is no hurrying her, and she takes no interest in her surroundings, and is fretful and irritable generally. On and off she was under treatment from March, 1898, and during 1899, missing a month occasionally, during four months only of the year 1900, during seven months of 1901, and during most of the present year up to date.

Present condition.—Her mental condition is much improved, especially while the treatment is continued, and indeed the patient is benefited in every way. The myxœdematous signs and symptoms are of course still present, but are not nearly so well-marked as they have been. I did not see her myself for the first three months of the present year, and then I noticed a great improvement in her appearance and in her mental condition when I again saw her in April. There is still a suspicion of fatty pads being present in the supra-clavicular regions, but they are with difficulty distinguishable; I cannot feel the thyroid gland at all.

Remarks.—Having regard to the early history of this patient and the appearances noticed when first seen, I suggest that this is a case of sporadic cretinism. The patient is now taking two and a half grains of thyroid-extract three times a day after meals; and it is proposed to increase this to ten grains a day. She is now attending a special school; her improvement is, of course, slow. If we can only have the treatment continued regularly it is hoped for still greater improvement in time to come. The improvement would, no doubt, have been much greater had the treatment been more systematically carried out, and at all events this case shows the importance of not leaving off the thyroid-extract.

(*Exhibited May the* 16*th*, 1902.)

Discussion.

Dr. G. E. Shuttleworth (Richmond) said that, having taken some interest in that class of case, especially from the educational point of view, when a medical examiner to the London School Board, it was to him very interesting to see the improvement which this child had made. He had seen similar cases before at the special instruction schools of the School Board. He thought the present patient might make even more improvement if the administration of the thyroid-extract were more regularly carried on. Mothers were often very remiss in the matter of giving the medicine, and therefore at the special schools an agreement was made by which the teacher regularly gave the medicine, of course under the doctor's orders. Recently it had become the custom to insist on every cretin child being submitted to treatment at a hospital, or under a private practitioner, as a condition of its admission to a special school for defective children, those whose mental defect was so slight as to enable them to be susceptible to education. If they were not subjected to thyroid treatment they were regarded as imbecile, and therefore not proper subjects for the special schools. Only that day he had seen a case illustrating the efficacy of thyroid treatment at a more mature age. Three years ago he was asked to see a gentleman forty years of age who was only thirty-six inches in height, and who had all the characteristic features of cretinism. He was a "chancery lunatic," and had been so for two or three years. It was only when one of the official medical visitors saw him that the idea of thyroid treatment in such a case was suggested. He (Dr. Shuttleworth) was asked to consult with the ordinary medical attendant about it. The man was put upon thyroid treatment, the doses at first being two and a half grains daily. When first seen there was little in the man to remind one of humanity at all; he looked like a squatting toad in the corner of his room, and stared into vacancy. He had a distortion of the chest and a deformity of the spine, and altogether was by no means a promising case. But treatment was persevered in, and he (Dr. Shuttleworth) was asked to see him three months later, the patient having been taken to stay at a farm. The report was that he had become more lively and even mischievous, and had taken to chasing the fowls. Thus the physical activity was considerably increased even after three months' treatment. Mentally also he had been improving up to the present time, as he was now able to converse in monosyllables and displayed considerable interest in his surroundings. That case seemed to show that it was never too late to try thyroid treatment, though, of course, no such striking results could be expected in adult life as in childhood.

Dr. Edmund Cautley asked whether in the case shown by Mr. Carre-Smith the thyroid gland altered after the cessation of the treatment. Occasionally it happened that after thyroid was given for some time and then stopped, the thyroid gland of the patient became enlarged, a subsidence subsequently occurring on the treatment being resumed. He agreed with the remarks of Dr. Shuttleworth, but in the case quoted by him he thought there would not be much further improvement.

The Chairman (**Dr. W. A. Wills**) said an interesting point in such cases was the large amount of thyroid which could be taken by children with that disease. It might be as large as seven and a half grains a day, which would be a very fair dose for even an adult case. It was often also noticed that the patient was the only subject of the condition in the family.

Mr. Herbert Carre-Smith, in reply, said he was very much interested in Dr. Shuttleworth's case. He could not pretend to any special experience in cases of the kind. He brought the case to show the marked differences in the condition while the patient was still under treatment. In reply to Dr. Cautley, he had not latterly noticed any alteration in the thyroid condition. The notes, which were taken four years ago, were not his own; but during the three years he had been seeing the case in the out-patient department there had been no change in the gland. But it was on the notes that in the first instance the gland could be felt, whereas it could not be now. His opinion had been supported by observers specially interested in the condition, such as Dr. Shuttleworth and Mr. Walter Edmunds. He agreed that the dose given in the present case was a large one, but it had been suggested that it should be increased as there were no untoward symptoms. It might be that a thyroid-tablet of a different manufacture might produce better results with less of the drug. He had impressed upon the mother the importance of the child attending a special school, and also of the medicine being taken quite regularly.

60. A CASE OF TUMOUR OF THE PERINÆUM.

By A. B. ROXBURGH, B.Ch.

The patient shown was a child, aged 2 years and 2 months, with a fatty growth occupying the left side of the perinæum and scrotum; the testicle was situated in the perinæum, and a hydrocele of the tunica vaginalis was present. The enlargement was noticed a few months after his birth, and had not increased in size.

(*Exhibited May the* 16*th*, 1902.)

61. A CASE OF VARICOSE VEINS WITH A CAVERNOUS NÆVUS.

By A. B. ROXBURGH, B.Ch.

The case was that of a female, aged 5 years, presenting varicosity of the veins of the left lower extremity, with an extensive nævus of the cavernous type on the outer side of the knee. A radiograph showed slight alteration in the length and shape of the tibia on the affected side.

(*Exhibited May the* 16*th*, 1902.)

Discussion.

Dr. Parkes Weber thought the presence of congenital varicose veins, or of developmental varicose veins, *i. e.* occurring in early childhood when associated with nævoid formation, was very interesting. He referred to a case shown before the Society by Dr. Hawthorne recently, when he considered—though Dr. Hawthorne did not—that the hypertrophied limb showed angiomata as well as dilatation of both arteries and veins. The tendency to venous varicosity varied in different people. In some that tendency was so great that the varicosity might be almost regarded as a kind of tumour formation. He thought every extreme could be met with, from varicosity produced only by continual venous distension to that which was obvious at or soon after birth. The present case illustrated the connection between nævoid tumour formation and the occurrence of varicose veins.

Mr. Clement Lucas said he had seen several cases similar to that shown, and saw no reason why the tumour should not be excised, and not only the veins but a large amount of cellular tissue also. He operated upon one case four or five times before he cured it, taking away portions of cellular tissue each time. There was a good deal of bleeding when he operated about the knee-joint.

Mr. A. B. Roxburgh, in reply, thought it would be well to modify the operation he had intended to perform after the remarks which had been made. He had intended to resect small portions of the varicose veins, and leave the nævoid condition undisturbed.

62. CASES OF ASYMMETRY OF THE SKULL.

By J. PORTER PARKINSON, M.D.

These are two children, aged respectively 2 years and 3 months, and 11 months.

In both cases the right side of the cranium is deformed, so that the frontal and parietal regions are indented; the extent can be judged from a drawing of the outline of the skull of one of them, obtained by the cyrtometer, and now exhibited. The drawing shows a marked depression, occupying the normal position of the parietal eminence, which is about three-fourths of an inch deep, and measures one inch in circumference.

A third child, recently dead, showed the same asymmetry, and the mother informs me that her husband and his mother have precisely the same shape of head.

The mother has had nine children, three of whom died shortly after birth; there were no miscarriages. All the births were easy, instruments having never been used.

The children seem intelligent, and have never had "fits" or any cerebral symptom. There are no other evidences of mal-development, and the palate is not high-arched.

I am informed that such asymmetry is not uncommonly seen in the inmates of asylums in cases of mental instability and degeneration, and a want of development of the corresponding cerebral hemisphere is usually found.

(*Exhibited May the* 16*th*, 1902.)

63. A CASE OF SUBLUXATION OF THE SHOULDER-JOINT.

By WALTER EDMUNDS, M.C.

The patient, a girl now 11 years of age, was first brought to the Evelina Hospital in 1891, when six weeks old. She was suffering from swelling and impairment of movement of the left shoulder, and also to a lesser degree of the left knee. Dr. Willcocks, under whose care she then was, appears to have regarded the case as one of specific epiphysitis; he treated the child with grey powder, under which treatment she recovered.

Attention has, however, recently been directed to the left shoulder by the inability of the child to raise the arm above the level of the shoulder when drilling at school; on this account she was again brought to the hospital.

On examination, it was found that the upper end of the humerus was small, and it is shown by skiagrams (kindly taken by Dr. Blacker), that the head of the bone has been arrested in development, while the great tuberosity is of normal size. There is no dislocation, but the joint is extremely lax. There is also creaking in the joint on its movement. The humerus at its lower end is wider than that of the opposite bone; it is probable that here also there has been some epiphysitis. The left humerus is one and a half inches shorter than the right. The muscles about the shoulder are normal, and the arm can be freely moved in any direction except upwards; it is a perfectly useful arm. The left knee is now quite normal.

The case is exhibited as showing the after effects of syphilitic epiphysitis. (*Exhibited May the* 16*th,* 1902.)

Discussion.

Mr. Clement Lucas said the case illustrated the value of X rays, and that he could quote a similar case which he was seeing from time to time. Soon after birth the child was noticed to be very delicate. He had operated upon the mother before her marriage on account of tuberculous glands of the neck. When a few weeks old the child had tuberculous otorrhœa. Some weeks later the child moved one hip stiffly, and as a result of that inflammation at the epiphysis the limb had become permanently shortened.

Mr. E. W. Clapham said he examined, for life assurance purposes, a man who had some sinking in of one side of his chest. He also appeared to have had an injury to his shoulder when a baby, and one arm was in consequence one and a half inches shorter than its fellow; but the man was in very good health, and was a good gymnast.

64. A CASE OF SARCOMA OF THE LEFT SUPRA-RENAL BODY IN A GIRL OF ELEVEN, WHO PRESENTED THE PHENOMENA OF "PRECOCIOUS PUBERTY."

By JAMES H. SEQUEIRA, M.D.

On the 13th of June, 1901, a very stout girl of 11 years of age, suffering from pain in the præcordial region and dyspnœa upon exertion, was admitted into the North-Eastern Hospital for Children under the care of the writer.

The child had had no previous illnesses, and her mother described her as being "thin and pale" until fifteen months before, when menstruation commenced. Since then she had been noticed to become very fat, and she constantly complained of feeling tired.

On admission, the appearance of the child was very striking. Her aspect was that of a stout little woman of about forty years old. She was 4 ft. 6 in. in height, and weighed 6 st. 3 lbs. The face was large, and the expression heavy and dull. The complexion was that of a brunette, but with no abnormal pigmentation of the skin. The skin everywhere was somewhat coarse, and there was a copious development of hair upon her chin and upper lip. The breasts were large, resembling those of a mature woman. The abdomen was distended with fluid, and the skin in its lower part showed numerous atrophic striæ, such as are seen in the pregnant condition, and with rapidly growing abdominal tumours. The pubic region was covered with long hair. There was some cyanosis, and the legs and abdominal wall were œdematous. The upper parts of the thighs showed atrophic lines in the skin similar to those upon the lower abdomen.

The thyroid gland could not be felt in consequence of the quantity of fat of the neck, but there was no excess of fat in the supra-clavicular regions.

The cardiac impulse was felt just outside the nipple line, and the area of dulness was increased to percussion. The first sound at the cardiac apex had a booming character, but there were no murmurs. The pulse-rate was 90 per minute, and the tension was definitely raised.

The respirations were 50 times per minute. The movements of the chest were somewhat laboured. Moist râles were heard all over both lungs.

The abdominal cavity was distended with a large quantity of free fluid, and in the left hypochondriac region a tumour, evidently the spleen, was palpable. This extended for four fingers' breadth below the costal margin. It was dull on percussion, but no notch could be felt. Behind the spleen another mass could be felt extending into and bulging the left loin. The actual shape of this second tumour, which appeared to be continuous with the spleen, could not be ascertained owing to the ascites. It was considered to be renal. The liver projected a little below the costal margin.

The urine passed in the first twenty-four hours was 44 oz. It contained albumen, one-fourth, on boiling. There was no sugar, and the deposit was free from blood and casts. The patient menstruated just before admission.

The bowels were freely opened by medicine, and this with the rest in bed brought about a rapid diminution in the œdema and ascites, and at the end of ten days the patient had lost nearly nine pounds in weight, and was more comfortable. She was able to sit up in bed, and amused herself by reading and needlework.

The urine passed averaged 28 oz. a day, and now contained a mere trace of albumen.

On the 30th of June signs of heart failure suddenly manifested themselves.

At 10 a.m. on the 1st July the pulse-rate was 144 to the minute, and the respirations 22 per minute. The temperature, which had hitherto been about normal, rose to 101° F. Stimulants and oxygen were administered, but failed to improve the condition, and the child gradually sank, and died at 10 a.m. on the 2nd July.

At the autopsy the subcutaneous fat was found to be over an inch in thickness over the thorax, and half an inch over the abdomen. The labia and lower abdominal wall were œdematous, as were also the lower extremities.

On opening the abdomen a large quantity of clear serum escaped, and the left hypochondriac and lumbar regions were found to be occupied by an enormous growth, which had pushed the spleen forward and the left kidney downwards. The tail of the pancreas lay in front of the tumour, and was not involved by it. The mass when removed weighed 3 lbs. 1 oz. It proved to be a small round-celled sarcoma of the left supra-renal body. The right supra-renal body was if anything slightly enlarged. The spleen weighed 5½ oz.; it was lobulated. The left kidney weighed 9 oz.; it was uniformly pale and somewhat tough, but the capsule stripped easily. The tumour was easily separated from the left kidney, which was free from secondary deposits, and had not been invaded by the growth. The right kidney weighed 8 oz.; it also was free from secondary deposits.

The liver weighed 4 lbs. 3½ oz. On section a small secondary nodule was found in the right lobe, and attached to the posterior border, close to the gastric impression, there was a mass the size of a hen's egg, of a similar nature to that in the left supra-renal body. It was apparently in connection with it, and may be considered as an extension of the primary growth.

The heart weighed 11 oz., the right ventricle and auricle were distended with ante-mortem clot. The wall of the left ventricle was slightly hypertrophied. The valves were quite healthy. There was a slight excess of fluid in the pericardium. The thyroid gland was normal.

There were no adhesions in the pleural cavities, but there was a slight excess of fluid in each. Both lungs were studded with secondary growths varying in size from a walnut to a pea. The stomach and intestines were normal.

The uterus was rather large, and the cervix and body were of about equal length. The ovaries were equal in size, and perhaps a little enlarged. On section several corpora lutea of menstruation were found, one of them being fairly recent.

Tumours of the supra-renal body are rare, and of twenty sarcomata, quoted by Affleck and Leith,* five were in children.

The earliest recorded appears to be that of Greenhow.† This was a sarcoma in a girl of twelve years old. It was three inches long and two and a half broad. There were no special symptoms.

A case described by Colcott Fox ‡ presents many features similar to my own. The patient was a girl aged two years. She had always been extremely fat. There was no pigmentation of the skin. The whole of the cutaneous surface was hairy, especially the genital and pubic regions. The abdomen had been enlarging for a year. The tumour was as large as a cocoa-nut, and weighed 1½ lbs. It was on the left side, and flattened the kidney. The liver weighed 38 oz. There was thrombosis of the inferior vena cava.

A case of Dickinson's (quoted by Fox), a child of three years old, had also an extraordinary development of hair, which had been noticed from the end of the first year. Here also there was no bronzing of the skin.

An enormous tumour of the left supra-renal body in a child of three and a half was shown at the Berliner Medicinische Gesellschaft, by Lazarus, in 1894. The growth weighed 12½ lbs. The child weighed 37 lbs. I can find no mention of abnormal hair development.§

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* 'Edin. Hosp. Reports,' vol. iv, p. 278.

† 'Path. Trans.,' 1867, vol. xviii, p. 260.

‡ 'Path. Trans.,' 1884–5, vol. xxxvi, p. 460.

§ 'Med. Press and Circ.,' May, 1894.

A supra-renal tumour occurring in a child of nine months old with numerous metastases in the cranium (causing exophthalmos), the kidneys, ovaries, and the liver is described by Cohn.*

The interest of my case lies in the early menstruation, the development of the breasts, and the growth of hair upon the pubes, the chin, and the upper lip. It is difficult, especially with the records of Fox's and Greenhow's cases before one, to believe that there was no connection between these phenomena and the tumour. It may, perhaps, be explained by the closeness of origin of the vascular supply of the supra-renal and that of the ovarian vessels, and the large size of the kidneys may be held to support this. The ovaries were not, however, enlarged.

The supra-renal body is, at any rate in part, developed in connection with the Wolffian body, and this latter structure takes part in the development of the genital apparatus, and, moreover, is closely related in its origin with the epidermis. A developmental connection, which, after all, is correlated with the development of the vascular supply, may be held to account for the association of the phenomena. It would be interesting to know if any of the cases of pelvic tumour, associated with "precocious puberty," have been instances of tumours arising in supra-renal "rests," which are not very uncommon in the neighbourhood of the ovary.

Another suggestion of the association of these phenomena appears to be the formation of an excess of an internal secretion from the supra-renal body. It would seem possible to put this to proof by the removal of the supra-renal bodies from young animals, and by a study of their subsequent development.

The author desires to acknowledge the assistance given him by Mr. Francis, formerly House-Physician at the North-Eastern Hospital for Children, in the careful notes made of the case. (*Read May the* 16*th*, 1902.)

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* 'Klin. med. Wochenschrift,' No. 11, 1894.

Discussion.

Mr. Clement Lucas thought very few would be able to refer to a similar case, as it was a very rare condition. The tumour was of great interest because it showed how many tumours situated in the neighbourhood of the kidney were diagnosed as renal when they were really supra-renal in origin. The chief interest in the case was the association of precocious puberty, and that was a subject which such a Society as this might take up and work out thoroughly The Society had not yet attempted to work out anything special in children's diseases by committees. In the first year it was proposed that a committee might work at subcutaneous nodules, but some thought the Society was then too young, and the idea was not carried out. Many cases of precocious puberty were associated with irritation of the ovary, but that did not seem to be so in the present case, though the tumour attacked parts not far removed in the matter of circulation and nerve supply. He thought a committee of the Society would be able to collect a good deal of new information on the subject. His experience was limited to one case, which he published in the Clinical Society's 'Transactions' many years ago. The subject was a red-haired child, seven years of age, who, when admitted into the Evelina Hospital, was menstruating regularly, had hair an inch in length on her pubes, and well-developed mammæ. He removed a solid tumour of the ovary, and with that the signs of precocious puberty gradually disappeared, but the pubic hair was not diminished while she remained under observation.

Dr. Parkes Weber asked what was the duration of the tumour, and at what age the precocity was first noticed. His idea was to try to establish a connection between the tumour and the precocious puberty. He thought it possible that the tumour had induced an excessive supply of blood to the neighbouring organs. Hofacker * said that out of sixty-two cases of precocious puberty three were found to have sarcoma of the ovary. The case was on record of a boy aged nine and a half years in whom development was altogether excessive; the right testicle was atrophied, but the left one was of enormous size. It was removed, and was found to contain an epithelial growth.† The boy then became more childlike, and his beard fell out. An analogy could be found in those cases in which a tumour of the thyroid gland had apparently induced excessive functional activity of the gland, and produced the symptoms of Graves's disease.

In a case of precocious menstruation in a girl aged six years

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* 'Centralbl. f. Gynäk.,' 1898, No. 41.

† E. Lacchi, in 'Riv. Ref. di Freniatria,' 1895, vol. xxi, p. 149.

Rein detected the presence of an ovarian cyst.* Brohl † mentions the case of a girl aged seven years with precocious menstruation. He removed a large cystic tumour of the left ovary, and there was no further menstruation as long as the child remained under observation, about two years. The question of precocious and delayed puberty is closely connected with the question of the causes of gigantism and infantilism, local and general. Moreover one form of gigantism, namely, acromegaly, is likewise associated with tumour formation.

Mr. H. B. Robinson said it would be wrong to hold the idea that precocious puberty must be necessarily associated with some tumour of the ovary or supra-renal body. A very remarkable series of cases came under his observation some years ago. Two children, one a fortnight old and the other two years old, were brought for him to say whether they were male or female. He said the elder was a female, but that the other should be christened some doubtful name so that it would do for either. Eighteen months later they were brought to him again, and then the elder one had developed a good deal of pubic hair. The younger one was then found to be a female, and to have also signs of hair on the pubes. Both these little girls had the clitoris much hypertrophied. A curious feature was that their brother, aged five years, had genitals and pubic hair as would be seen in an adult.

Dr. George Carpenter said that, so far as he could judge, the ovaries and Fallopian tubes in the specimen shown were perfectly natural in size. He remembered Mr. Lucas's case, and his impression was that the pubic and axillary hair disappeared from the child after the operation. All were acquainted with diseased ovaries and Fallopian tubes in young children. He had seen several cases of tuberculous disease of the ovaries and Fallopian tubes without evidence of precocious puberty. One child had cystic disease of the ovary, about the size of a Tangerine orange, when it was twenty-two months old. There was no evidence of precocity; it was operated upon successfully, and was now quite well. He had seen a sarcomatous ovary in a child of three and a half years old associated with other sarcomatous abnormal deposits and infiltration of both kidneys without precocious puberty. Precocious puberty was not necessarily therefore associated with even extensive disease of the ovaries and Fallopian tubes. He had not seen a case of precocious puberty in connection with diseased states of the uterus in children. He believed there were cases on record of a flow of blood from the female genitalia in cases of sarcoma of the vagina and uterus. He was acquainted with the opposite condition, viz. infantilism in association with atrophied testicles secondary to congenital syphilitic orchitis. It was outside his experience to find it in association with renal or supra-renal sarcomata, unilateral or bilateral, or with retro-perineal or abdominal sarcomata, although not a few such cases had passed through his hands.

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* Vratch, 1894.

† 'Centralbl. f. Gynäk.,' 1897, No. 4.

The Chairman (**Dr. W. A. Wills**) said that in an article on the subject in Allbutt's 'System of Medicine' precocious puberty was mentioned as being associated with malignant disease of the kidney, and it was the only form of kidney tumour with which precocious puberty seemed to be associated; but no reference was given to the case.

Dr. J. H. Sequeira, in reply, said the history was that the child had never had any illness until fifteen months previous to her admission into the hospital. Up to that time she had been thin and pale, and at that date the catamenia started. The mother dated the increased size of the abdomen from the commencement of that function.

65. A NOTE ON A CASE OF KERATOMALACIA.

BY SYDNEY STEPHENSON, C.M.

So far as my experience extends, this peculiar disease of the cornea occurs exclusively in children under one year old. It is distinctly rare. Its victims suffer from a marked degree of athrepsia, which may be due to several causes, of which the most important undoubtedly are tubercle, syphilis, and imperfect feeding. It is a significant fact that in the few cases that have been examined at this hospital (the North-Eastern) post mortem, disseminated tubercle was found in all.

The ocular symptoms of keratomalacia are distinctive. A more or less central ulceration of the cornea is seen, and this is associated with few if any of the signs which usually accompany such a process, namely, redness of the eye, lacrymation, photophobia, or spasm of the eyelids. The eye gives one the impression of being curiously dry, as it were. There is no conjunctivitis. The little patient, who is often reduced to mere skin and bone, resents hardly at all attempts to examine his eyes; he lies perfectly placid, whining occasionally, but does not cry or evince any other sign of discomfort. It is, in my experience, the exception rather than the rule to find triangular, white, greasy-looking patches on either side of the cornea. Still, considerable stress has been laid upon these curious products by several writers on the subject, and, when present, they are certainly characteristic.

At a later stage the ulcer of the cornea becomes purulent, and pus is likely to be found in the anterior chamber. The cornea itself perforates, and some of the contents of the eyeball, as the crystalline lens, escape. Finally, the globe becomes shrunken and atrophic, being eventually represented (if the patient live long enough) by a small, pallid nodule lying in the depths of the orbit. The malady, as a rule, attacks both eyes.

The disease is evidently due to a pus-infection of an imperfectly nourished cornea, perhaps following some slight almost inappreciable traumatism. It seems unlikely that a specific micro-organism exists. At various times, I have found almost all the common pyogenic organisms in scrapings and cultivations from the corneal ulcers. The xerosis bacillus is frequently seen, but has almost certainly nothing to do with the disease.

Prognosis, as regards both the eyes and life itself, is bad. Nevertheless, I have more than once seen the eye disease arrested in cases where the patient survived.

It would, in my opinion, be a fair generalisation to say that ulcers of the cornea, especially if bilateral, in infants between the ages of two and twelve months are far more likely to be due to keratomalacia than to any other disease. Such patients are too old for the corneal complications of gonorrhœal ophthalmia, and almost too young to suffer from the other great cause of corneal ulceration in childhood, *viz.* eczematous keratitis.

Diagnosis.—The diagnosis of keratomalacia should offer no difficulty to one who is aware of the existence of the disease. Still, two conditions, both of which occur in childhood, might be confounded with it, namely, (1) keratitis xerotica and (2) trophoneurotic keratitis. *Keratitis xerotica* (Feuer) is liable to occur in children who lie for long in a state of so-called coma vigil, from whatever cause. The semi-closed eyelids allow the lower third of the cornea to be exposed to the air. Accordingly that portion of the cornea becomes dry, and this, together with the serious general condition of the patient, allows it to fall a ready prey to any pyogenic organisms. An ulcer, shallow at first but soon becoming deeper, forms near the lower margin of the cornea, and, if the child lives long enough, this becomes complicated with deeper changes, which may lead to loss of the eye. The affection, so far as I have seen, is invariably bilateral. It is sometimes called "Keratitis e Lagophthalmo" (Fuchs). *Trophoneurotic keratitis*, a rare and painless disease, is caused by paralysis of the fifth cranial nerve. It gives rise to a very peculiar appearance of the cornea, since practically the entire anterior epithelium of that structure is cast off, except perhaps for a narrow rim at the extreme periphery. In its further course the cornea becomes infiltrated and cloudy, pus appears in the anterior chamber, and the eye is lost by suppuration, or at best is left with a corneal cicatrix in which the prolapsed iris has become entangled. There is anæsthesia of the upper and lower eyelid on the affected side.

The following case is fairly typical of keratomalacia.

Ernest B—, aged 4 months, attended my clinic for the first time on February the 26th, 1902, suffering from ulceration of the corneæ. He was a full-time child, and is said to have been well-nourished at birth. A few days after birth he refused to suck, and since then has been fed with great difficulty on cow's milk and barley-water. There was no history of ophthalmia neonatorum. No "snuffles," rashes, or other signs of inherited syphilis were detected. The child was stated to have had a "fit" on the eighth day after its birth. He is the youngest of four children, of whom the other three are said to be healthy. The eyes have been bad for about one month.

On admission.—He is a puny, apathetic, wasted baby, weighing in the nude 8½ lbs. only. There is no evidence of syphilis. The temperature is 97·4° F. *Right eye.*—A small infiltration lies towards the centre of the cornea, and stains faintly when treated with fluoresceïn. There is no vascularity of the eye. The tension is normal. *Left eye.*—A large and dense infiltration, which stains deeply with fluoresceïn, covers the centre of the cornea. There is merely a trace of ciliary redness, and the eye looks dry, as it were. There are no changes in the palpebral conjunctiva, and the tear passages are natural. The tension is normal. The eyes can be examined without the baby evincing the least sign of discomfort, and its silence and apathy are striking features of the case.

Progress (March the 5th).—The child has lost one pound in weight during the week. The temperature has not risen above 99·8° F. The bowels are usually opened three times daily, but sometimes not at all. *Right eye.*—The ulcer has healed, and is now replaced by a small more or less central nebula. *Left eye.*—There is trifling dusky ciliary redness. The entire pupil is hidden behind a purulent ulcer of the cornea. Pus can be recognised towards the outer side of the anterior chamber. There are no physical signs in the chest or the abdomen.

9th.—The whole of the left cornea is now involved by purulent infiltration, and the cornea is on the point of perforating. Later in the day the cornea did actually perforate, and the crystalline lens escaped from the eye.

12th.—The left eyeball is in a condition of panophthalmitis, and is somewhat proptosed.

April 2nd.—The left eyeball is now shrunken, and the signs of inflammation have almost disappeared.

May 7th.—The left eye is now represented by a small nodule, free from congestion, lying in the orbit. A button of granulation tissue exists in the former site of the cornea. I detected the existence of physical signs in the baby's chest, etc., and accordingly requested my colleague, Dr. J. H. Sequeira, to examine the case. His report ran as follows:—"There is evident consolidation of the left upper lobe, with breaking down of the lung. There are enlarged glands in the mesentery, and the spleen also is enlarged, being easily palpable a finger's breadth below the costal margin. I consider the lung and glands certainly, and possibly also the splenic trouble, as tuberculous."

During the three months the child remained in the hospital the successive weights were 8½ lbs., 7½ lbs., 7 lbs., 6¾ lbs., 7 lbs., 6¾ lbs., 7 lbs., and 6¼ lbs.

The baby died on May the 17th, 1902, and, to my great regret, permission for a post-mortem examination could not be obtained. (*Read May the* 16*th*, 1902.)

Discussion.

Dr. Edmund Cautley said he hoped Mr. Stephenson would subsequently add the post-mortem notes of the case. He did not wish to throw any doubt on the diagnosis, though the temperature chart was not like that of tuberculosis. Still, the chart of tuberculosis in children varied a good deal. He asked whether the gonococcus could be excluded as the cause. Even though ophthalmia neonatorum could be excluded, he did not think the gonococcus could be.

Mr. Sydney Stephenson, in reply, said the out-patient sheet was marked with the following note: "No ophthalmia neonatorum;" so there had been no history of infantile ophthalmia in the case. Moreover, the symptoms did not begin until the third month, whereas ophthalmia neonatorum was much more likely to begin on the third day. The symptoms were those of keratitis, and not of conjunctivitis. Clinically, therefore, the gonococcus could be excluded.

66. CASE OF DEFORMITY OF RIGHT SHOULDER (? SPRENGEL'S DEFORMITY).

BY H. BETHAM ROBINSON, M.S.

THE little girl, aged 11 years, in whom the above was noted, was first brought to me at the East-London Hospital for Children some six weeks ago. She had always had good health, and the deformity had only been noticed two months. In the erect position the right shoulder is noticed to be considerably raised. With reference to the scapula itself, its inferior angle is a full one and a half inches above the other side and rather nearer to the middle line. The superior angle, on the other hand, is a greater distance from it, and can be felt under the enlarged muscles. The length of the right vertebral border is a quarter of an inch shorter. The right coracoid process is more prominent than the one on the left side. There is no "winging" of the scapula. The muscles generally are well developed, and all the movements fair except that of elevation of the limb vertically. The upper part of the trapezius muscle (and ? also the levator anguli scapulæ) is contracted. The spinal column shows some "lateral curvature." The right anterior superior spine is a quarter of an inch below and is in front of that on the left side; the right side of the belly is more prominent. The ribs on the right side are spread out. In short, there is a long curve with its convexity directed towards the right side of the body.

(*Exhibited May the* 16*th,* 1902.)

Discussion.

Dr. Travers Smith said that last year he showed at the Polyclinic a case which was very similar. Sprengel's theory was that *in utero* the arm became fixed behind the back of the neck, and the shoulder was raised thereby, thus bringing about the deformity.

Dr. C. O. Hawthorne thought that in some of the cases a bony process had been found passing from the spinal column towards the upper part of the scapula, and he would like to hear whether that point had been investigated in the present case.

Mr. H. B. Robinson, in reply, said he had omitted to point out a certain thickening about the superior angle of the scapula. But though bossiness could be felt there, no process of bone seemed to run towards the spinal column.

67. NOTE ON A CASE OF ARTHRITIS ACCOMPANYING OPHTHALMIA NEONATORUM.

By C. O. HAWTHORNE, M.D.

For a recognition of the fact that the purulent ophthalmia of newly-born infants may be associated with joint complications analogous to those which not infrequently attend urethritis we are indebted to Mr. R. Clement Lucas.* His first observation, made so far back as 1885, was perhaps not unnaturally subjected to the ordeal of a somewhat incredulous criticism. From this, however, it emerged successfully, and the suggestions that his case might have been one of epiphysitis, or of syphilitic synovitis were successfully met by clinical arguments that can hardly be gainsaid. The lapse of time has provided further evidence. This is by no means abundant so far as its volume is concerned, but it is supplied by a number of independent observers and contains elements which raise it to the level of established proof. To judge from systematic works dealing with the subject of ophthalmia neonatorum it would appear that Mr. Lucas's original observation hardly attracted the attention which it deserved, though for this some excuse may perhaps be found in the fact that, with one exception, recorded by Mr. R. G. Fendick,† also in 1885, no parallel case was recorded in Great Britain during a period of thirteen years. And when Mr. Lucas, in 1899, introduced the subject to the Royal Medical and Chirurgical Society a search through European medical literature had resulted in the discovery of only twenty-three cases. Writers on gonorrhœal rheumatism subsequently to Mr. Lucas's case, however, began to recognise the occasional occurrence of joint complications in ophthalmia neonatorum, and the association was mentioned by Dr. Archibald E. Garrod * in 1890, and by Dr. J. Mitchell Bruce † in 1894. More recently Mr. Sydney Stephenson ‡ has given some interesting particulars of the character of the joint affections, and Dr. L. Vernon-Jones § has also drawn attention to them. Yet, from what has been stated above, the cases are anything but numerous, and so it seems desirable that examples of the condition should be put upon record, more particularly as in some instances the subacute character of the inflammation and its temporary duration may cause it to be overlooked.

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* 'Brit. Med. Journ.,' 1885, vol. i, p. 429; vol. ii, pp. 57, 699, 773; 'Transactions' of the Royal Medical and Chirurgical Society, vol. lxxxii, p. 137.

† 'Brit. Med. Journ.,' 1885, vol. ii, p. 830.

The example now to be reported can only claim the interest that it confirms the results of the earlier observations, more especially as regards the possibly quiet nature of the arthritis, and the prompt and thorough restoration of the efficiency of the affected joints. The diagnosis in this individual case was not confirmed by bacteriological examination, though this has been accomplished in other instances in reference both to the conjunctival discharge and the synovial effusion. But as we have medical warrant that the father of the patient had gonorrhœa two months before the child was born, that the mother was subsequently affected, and that the child began to suffer from purulent ophthalmia when two days old, the diagnosis is not open to doubt.

The earlier particulars of the case, for which I am

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* 'Treatise on Rheumatism,' p. 185 (Charles Griffin and Co.).

† Quain's 'Dictionary of Medicine,' second edition, vol. ii, p. 681.

‡ 'Contagious Ophthalmia,' 1890 (Baillière, Tindall, and Cox).

§ 'Gonorrhœal Arthritis,' 1891 (H. K. Lewis).

indebted to Dr. John Clarke, are as follows:—The affection of the eyes commenced two days after birth, and an abundant purulent discharge continued for fully three weeks. When the child was a fortnight old the right shoulder-joint became considerably, and the right wrist slightly, swollen, without apparently causing the child much discomfort, and the swelling subsided in the course of a fortnight without any very active treatment. When the child came under my observation he was six weeks old. There was still a slight amount of muco-purulent discharge from the conjunctivæ, and the right cornea presented a small abrasion, from which a diffuse haze spread through the corneal substance. It may, perhaps, be conjectured that here is to be found the explanation of a further outbreak of joint inflammation. For while the joints which were first attacked, that is the right shoulder and wrist, had completely regained their normal characters, the left elbow-joint was much enlarged, and the swelling extended down the radial side of the forearm for a considerable distance, giving here a sensation of deep fluctuation, as though the sheaths of the muscles were involved. The skin was not reddened, and though the child objected to manipulation of the joint, he appeared otherwise to be free from pain, and his general nutrition was entirely satisfactory. It was decided to fix the limb and to continue the local treatment of the ophthalmia, and under these measures the patient made a rapid recovery, so that in from two to three weeks the elbow-joint and forearm had regained in every respect their normal characters.

Attention may first be drawn to the subacute character of the successive attacks of arthritis. Almost from the outset of his observations Mr. Clement Lucas recognised that two varieties of arthritis associated with ophthalmia neonatorum occurred, viz. (1) a very acute form with features suggesting a tendency to suppuration, and (2) a milder form accompanied by a great deal of effusion and pain on movement, but with little or no surface redness. The present case adapts itself exactly to this latter form, A second feature worthy of note is the extension of the swelling beyond the limits of the joint cavity. This is, of course, a well-recognised event in many cases of arthritis of urethral origin, and Mr. Sydney Stephenson has drawn attention to "effusion into the sheaths of the tendons" in the neighbourhood of the affected joints in cases where the primary gonococcal inflammation involves the conjunctiva. The joints most commonly affected would appear to be the knees and the wrists, but in the present case the knees escaped altogether, and the stress of the disease fell on the right shoulder and the left elbow. It may be added that no history of injury could be obtained, and there was no evidence of syphilis in either the child or the parents. Lastly, the promptness and completeness of the restoration of the joints to the normal may be noted. This is in harmony with other records, and supports and justifies the presentation of a favourable prognosis in these cases. It is a feature of the condition in pleasing contrast to the obstinate and often prolonged evil effects which so frequently attend the usual form of gonorrhœal arthritis. The explanation is doubtless to be found in the ease and readiness with which a purulent ophthalmia can be successfully treated as compared with the difficulty of bringing to a close the opportunities for absorption of a purulent discharge offered by the urethra and its related glandular recesses. The difference between the course of the arthritis in a case of gonorrhœa on the one hand, and in ophthalmia neonatorum on the other, lends emphasis to the statement that the checking of the urethral or vaginal discharge is the first claim in a case of gonorrhœal arthritis. It can hardly be supposed that the delicate tissues of the child's joints are more resistent to the gonococcal poison than are those of the adolescent or adult, and the explanation of the contrasted joint complications must therefore be found in the readiness with which the supply of poison is removed in the one case, and the difficulty with which it is checked in the other.

(*Read May the* 16*th*, 1902.)

Discussion.

Mr. Clement Lucas thanked Dr. Hawthorne for the very excellent report of the case, and for his kind reference to his (Mr. Lucas's) own work. It was a pleasure to him to know that an observation he made clinically many years ago, when the present means of bacteriological examination were not available, had proved to be absolutely true, for at that time the gonococcus was only recognised by very few people. He also expressed his gratitude to Mr. Sydney Stephenson, who appeared to be the first in this country to afterwards allude to Mr. Lucas's observation in a lecture which he gave at the Evelina Hospital for Children. The first case, Mr. Lucas observed, was a very acute one, accompanied with such intense redness, pain, and œdema that the joint seemed as if it must suppurate. The fact that it did not attracted his attention, and it seemed to be like the condition met with in acute gonorrhœal rheumatism in adults, especially in women. He predicted that, as the first case was proved to be an acute gonococcal joint disease, there must be a subacute form similar to that which Dr. Hawthorne had so exactly described. Mr. Lucas's second case illustrated the subacute form. With regard to recovery, it seemed to be almost always complete, even though the acuteness of the inflammation was very great. Very few joints had suppurated, and in those another organism was generally found associated with the gonococcus. He was sceptical about the gonococcus alone being able to cause suppuration, and it was easy to understand how another organism could gain access to the urethra. There was otorrhœa in one case that suppurated. The peculiarities of the affection were the tendency to recovery without suppuration, and the association of purulent ophthalmia.

Mr. Sydney Stephenson said he had seen altogether five cases of what might be called Lucas's joint disease. He referred to the subacute form of arthritis associated with ophthalmia neonatorum. He had not yet seen a case with acute symptoms, such as was first described by Mr. Lucas in the 'British Medical Journal' many years ago. All his (Mr. Stephenson's) cases, with one exception, had ended favourably, and the only treatment adopted was that directed to cure the ophthalmia, as Mr. Lucas had mentioned in a paper read before one of the societies. The exceptional case was one which Mr. Stephenson saw in association with Mr. Douglas Drew. The child first came under his care for ophthalmia neonatorum, and in the third week, when the ophthalmia was recovering, one of the knee-joints became inflamed. The case then passed into Mr. Drew's hands, who aspirated the joint, but without being able to find gonococci. That knee went to the bad, and was now ankylosed. With reference to Dr. Hawthorne's interesting case of course there was a fallacy, though apparently only an unimportant one. The discharge from the eyes was not examined for gonococci, but as there was well-ascertained gonorrhœa in the father and mother, and the onset in the child was on the second or third day of life, the apparent logical lapse could be forgiven. He suggested to Dr. Hawthorne that in a similar case it would be worth while to examine the scanty conjunctival discharge. Mr. Stephenson's experience was that gonococci persisted, despite proper treatment, for weeks or months, and even if the child were seen in the second month it was not too late to find gonococci in the eye discharge.

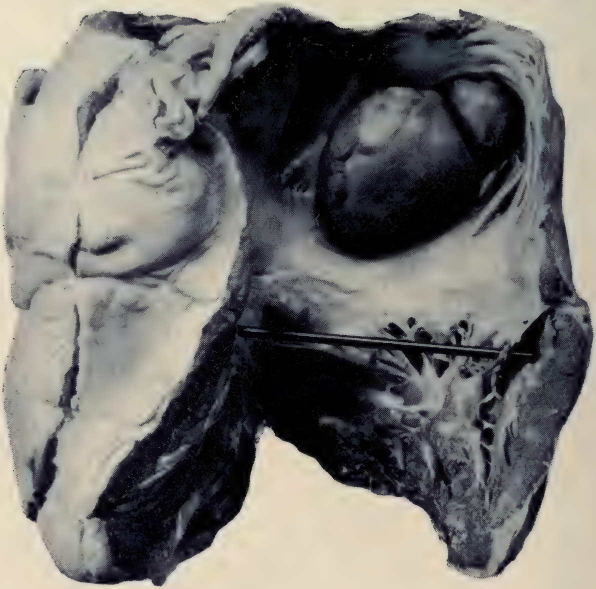
Dr. C. O. Hawthorne, in reply, admitted that it was unfortunate that the discharge was not examined, but there could be no reasonable doubt as to the nature of the case. The joint complication was similar to that first described by Mr. Lucas. He deprecated strongly any heroic local investigation of the joint with a view to verify the diagnosis. That appeal was strengthened by the experience which Mr. Stephenson had quoted. Although there was nothing new in the case, he thought it would be useful to present his notes of it, and he was glad he had done so, if only because it had been the means of eliciting from Mr. Lucas the very interesting remarks he had made.

68. AN ORGANISED INTRA-CARDIAC THROMBUS.

By J. PORTER PARKINSON, M.D.

This specimen was taken from a boy, aged 8 years, who has been under observation at the North-Eastern Hospital for Children since January 1901.

There was a history of scarlet fever in 1889, since when he had suffered from œdema of the face and legs, enlargement of the abdomen, and dyspnœa. These signs were noted on his first admission to the hospital, when he also had orthopnœa. The heart was enlarged upwards and to the right, but the apex-beat was in the normal position, and the heart-sounds were sometimes normal, but at other times a presystolic murmur was heard at the apex. There were signs of bronchitis and of some consolidation at the base of the left lung. The abdomen was enlarged,



Dr. J. P. Parkinson's Case of an Organised Intra-cardiac Thrombus.

Adlard & Son, Imp.

but there was no sign of fluid. The liver was enlarged and rather hard; the urine was scanty, and contained a cloud of albumen.

He improved for a time, but in July he was readmitted into the hospital with similar signs as before, but in addition he was rather cyanotic; there was some ascites, and the amount of albumen in the urine had increased, and a few hyaline casts were present. The liver was larger and reached to the level of the umbilicus. The heart's apex-beat had moved to well outside the nipple line, but no murmurs were heard. The ascites increased, and occasional paracentesis was necessary at intervals of about two months duration till death occurred suddenly in April, 1902.

During the last few months of life the subcutaneous œdema entirely disappeared, and the limbs were cold and blue.

At the post-mortem examination the lungs were in a condition of brown induration with some recent patches of pneumonia. There was a considerable amount of ascitic fluid; the liver was very large, and nutmeg in character; the spleen was small and indurated. The kidneys were normal in size, but exceedingly tough.

The pericardium was not adherent; the heart weighed a little over five ounces, the left ventricle was hypertrophied and the other chambers much dilated and hypertrophied. The valves were normal, the tricuspid and mitral orifices easily admitted the thumb. At the junction of the auricular appendix with the right auricle was seen a rounded mass the size of a walnut, which was attached by a short pedicle to the inferior part of the auricular wall, and close to it was a similar pedunculated mass the size of a hazel-nut, while in the appendix was a third the size of a pea (*vide* Plate VII). In the recent state these masses were hard and apparently consisted of organised fibrin, except in one part of the largest one, where there was a cavity filled with broken-down blood-clot. The surface of the masses was smooth and shining.

Such cases of organised thrombi in the heart appear to be very rare, softer white coagula being much more common. Dr. Ogle in 1863 reported a series of seven cases to the Pathological Society; these with one exception occurred in cases of chronic kidney disease, and their situation was in the right auricle, as in the specimen before the Society; but his cases all occurred in adults, and the thrombi were not of such old date as is this specimen.

A case of vascularised intra-cardiac thrombus was shown by Dr. Voelcker before the Pathological Society in 1893; this was about the size and shape of a champagne cork. It was attached to the margin of the foramen ovale, projected into the left auricle and fitted over the mitral orifice. In this thrombus embryonic vessels were seen.

Dr. Ewart and Dr. Rolleston showed before the Clinical Society in 1897 a case of an organised thrombus, which was attached to the wall of the left auricle and gave rise to a presystolic murmur during life. This apparently dated from an attack of pneumonia a year earlier.

It seems possible that in the present specimen the thrombi date from the attack of scarlet fever three years ago, and they all appear to be about the same age.

(*Exhibited May the* 16*th*, 1902.)

69. LIST OF CASES SHOWN BY DR. HENRY ASHBY AT THE CHILDREN'S HOSPITAL, PENDLEBURY, MANCHESTER.*

1. A case of wasting of the plantar muscles of both feet producing pes cavus and condition of hammer-toe in each great toe. In addition there was some wasting of the calf muscles and of those of the thighs.

2. A case of pseudo-hypertrophic muscular paralysis.

3. A case of a curious spastic condition of the lower limbs induced by any voluntary movement.

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* From 2.30 to 3.30 p.m., prior to the formal opening of proceedings of the Provincial Meeting on June the 21st, 1902.

4. A case of cerebral tumour. Vomiting occurred almost daily. There was paralysis of the right sixth cranial nerve; paresis of the left hand and arm; and paresis of the left leg.

5. A case of obstetrical paralysis in which the fifth spinal nerve of the right side was chiefly implicated, so that the rhomboid and the supra- and infra-spinatus muscles were chiefly affected. The child had improved since it was first seen.

6. A case of obstetrical paralysis in which the fifth spinal nerves of both sides were affected.

7. A case of Hodgkin's disease.

8. A case of congenital syphilis with an enlarged liver, and the tibiæ and the bones of the forearm were curved and hypertrophic.

9. A case of congenital syphilis with signs of old keratitis. There were rhagades and the lower limbs were covered with punched-out scars of old ulcers. Curious trophic lesions occurred on the toes, which constantly broke down.

10. A case of mitral disease associated with apical systolic and diastolic murmurs.

11. A case of mitral regurgitation which had developed pericarditis. Double friction-sounds and a systolic mitral murmur could be heard.

12. A case of aortic disease associated with systolic and diastolic aortic murmurs.

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70. SOME REMARKS ON HEART DISEASE IN CHILDREN.

By A. E. SANSOM, M.D.

In the first place I have not a paper to read; I have only a few remarks to make. Some of us are accustomed to a "few remarks," and they may be pleasant, or they may be otherwise. But I have a very agreeable task in the first place, and that is to thank the Authorities of this Hospital,* especially our Chairman on this occasion—whom we all revere, as well as for whom we have a very great affection,—for their goodness to us in allowing us to come here and see the wealth of material which they have. It is all very well to make apologies for the wards, but it seems to me there is no place in the whole country, or any other country, I think I may say, in which disease in children is better to be observed and studied than here. And here let me say that, on the other hand, this Society is not only honoured, but it honours the place. It is founded upon the best principles, I believe, and I think the title is worthy of all commendation. It is a Society for the *study* of disease in children, and it is upon those lines that I have to make a very few remarks,—that is to say, to read the title as it was suggested to myself, "Some Remarks on Heart Disease in Children." When we look over the great realm of children's diseases, and when we narrow our view a little bit to the question of heart affections in children, I should say that in the enormous proportion it is the rheumatic heart which we have to deal with. It is quite true to say that in children especially we have to consider anomalies, malformations which we do not meet with so often in the adult, because life, unfortunately, is not preserved long enough; but even when we do, we find that in a large number of cases, when we study them as Dr. Fisher has done on the post-mortem table, there is not infrequently superadded to it a form of disease essentially of rheumatic type, that is so far as we have morbid anatomy to guide us. And that is no original observation. It was made by Dr. Peacock in the very early periods of investigation on the subject. So that even when we have malformations to consider we have also the superaddition of rheumatic troubles to think of also. And then we have to think in any case whether the question of congenital anomaly comes in—whether the congenital, or seemingly congenital anomaly is due to intra-uterine endocarditis, or whether it is due to congenital malformation. Now there is such a thing as congenital endocarditis; that I have proved myself, and I know many others have clearly proved it also; and it always becomes us, therefore, to question, in any case that seems to be one of congenital anomaly, whether also there is not a question of congenital endocarditis, producing what seems to be a congenital anomaly.

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* The Children's Hospital, Pendlebury, Manchester.

In this communication I only propose to consider the following groups of what I will call the chief condition of heart disease in children, and that is the rheumatic heart. The congenital anomalies I shall not consider at all; they are too rare for my present subject, and I shall only consider the more common forms of heart disease in children. And those more common forms I would concentrate as examples of the rheumatic heart.

As to the rheumatic heart, the groups, varieties, and phases which I propose to consider are, first, the temporarily swollen or enlarged heart of rheumatism; second, the heart of rheumatic pericarditis (these are only very briefly put); third, the heart of rheumatic endocarditis with ulcerating valvular disease, severe or extensive; fourth, the heart of slow, insidious endocarditis inducing mitral stenosis. By *rheumatic* I mean a morbid condition in strict relation with my conception of the rheumatic process, the strict phenomena of rheumatism. I do not mean any specially painful implication of the joints as an indication of this condition. It may be strange to assume that rheumatism is not essentially a painful disease, but this is my deliberate conviction. The arthritis of true rheumatism is certainly very painful in some cases. But, painful while it lasts, it is singularly controlled and influenced for good by medicines—salicine and salicylates, —and seldom lasts longer than a fortnight. The pain and swelling of the joints disappear, leaving no permanent morbid changes, though there may be, and often are, waves or storms of recurrence. There are many cases of arthritis, however, that are not rheumatic. I am of opinion that osteo-arthritis, so-called rheumatic gout, is dependent in many cases upon infections which have no relation with rheumatism; that the morbid changes in this disease are more pronounced and more lasting, the pains are much more severe and protracted, and the resulting deformities much more significant than in the case of true rheumatism. Now here I must just call your attention to somewhat of a rarity, that is to say, osteo-arthritis in children, and the lessons that I think it teaches us. It may be remembered, and you will see it in the last volume of the 'Reports' of this Society, that I brought forward a case which I called one of osteo-arthritis in a child. The boy was aged 11 years; it does not occur generally at a very early age. Nearly all the large joints were swollen, and it was very long-lasting—six months under our observation. There was a little improvement under massage and so on, but not a great deal; but it did not comport itself in the way that I think is usual with acute rheumatism generally. Acute rheumatism in the child is evidenced mostly by rise of temperature, by painfulness, which may be very slightly marked, in a few joints, by anæmia, by skin troubles, especially erythema, or by peliosis rheumatica, as it is called,—that is purpura,—and sometimes by urticaria. Other forms of erythema, like erythema nodosum, are very doubtfully associated with rheumatism. I do not think erythema nodosum has anything to do with rheumatism. But at any rate it is marked by some skin affections and by rises of temperature. But in childhood, as I need not say to members of this Society, who have plenty of opportunities of investigation, the phenomena, but not the complications of rheumatism are very much to the fore. There may be pericarditis, there may be endocarditis, and there may be the swollen heart of rheumatism, without any very marked phenomena to show that there are arthritic phenomena at all. Therefore it comes to be a question—and, unfortunately, we are very often able to trace it by morbid anatomy—of these things occurring with very little symptoms. And they exemplify a form of rheumatism, a very marked phase of the rheumatic process in the child, without the intervention of arthritis to interpret it as rheumatic. Therefore it does not do for us to take arthritis as an index of rheumatism.

There are other things which come before us markedly in the rheumatism of the child. To come back to this case which I spoke of and its lessons, there were very marked painfulnesses, lasting for at least six months, very marked deformities, and twelve months afterwards I had an opportunity of seeing the child again; it had been under my care for a considerable time. The characteristic lessons are these: that the arthriticism—the arthritic conditions—are much less; the wasting conditions are very marked indeed. So marked is the wasting of the muscles, and the deformity which results from that wasting, that I have been able to hang thereupon some remarks to my clinical-clerks and house-physicians. And I say that in some of these cases of arthritis—and I say it with plenty of opportunity for correction, and I know you will correct me if I am not right—the arthritis is not rheumatic. He has no cardiac lesion and no approach to it. In the form of arthritis which I speak of, which is not rheumatic according to the best of my knowledge and belief, sometimes the arthritis is more than the nerve phenomena, and sometimes the nerve phenomena are more than the joint phenomena, and it is a question, very often, of the preponderance of the one or of the other. In the rheumatic osteo-arthritis, the so-called rheumatic gout of children, there may be very much destruction or alteration of joint, or there may be very little alteration of joint and a very great deal of wasting owing to nerve causes. I have had some photos. and radiographs taken in this case, and they will compare with those in a former volume of the 'Reports.' I think the radiographs show that the chief signs are in the feet, and that the swellings which were about the joints were very much less; whereas the deformity and wasting are still very marked indeed, although it is less in the hands than it was previously.

To come then to our former topic, the rheumatic heart of childhood, this is essentially a rheumatic phenomenon, and it very often is not to be interpreted by any form of arthritis at all. The heart of osteo-arthritis is a very irritable heart, very often inclined to be quickened, sometimes inclined to be slow, sometimes irregular; but it is not associated with any organic disease of so-called rheumatic type.

And now you may say I have been labouring rather to show that the common notion of taking arthritis and painfulnesses and painful joints as a sign of rheumatism is not a justifiable thing, and that we who study disease in children are especially disposed to combat that usual notion so prevalent amongst the public, and I may say those members of our own profession whose practice does not lie so very much amongst children. Rheumatism is not essentially a joint process, it is only adventitiously a joint process.

I will now briefly refer to my first group which I have mentioned of the truly rheumatic heart in children; and I may say that so far as my own experience goes it entirely endorses the observations of my friends Dr. Lees and Dr. Poynton, who have shown that in child rheumatism, as well as in the rheumatic fever of adults, the heart may be temporarily enlarged. Now I have frequently found that the case in the wards, and I have demonstrated it. I do not think you want any severe and unusual processes to demonstrate that; your own fingers are the best. The first thing to find is, where is the pulsation of the heart to be felt? If that is felt outside the normal, to the right or the left, you make a note of it. Then I agree with Dr. Lees that when you have found the point—I do not like the phrase "diffuse beating," because it is often felt in more than one interspace,—when you feel it you can reckon that the ventricle extends at least a finger's breadth outside that, you may say the heart is enlarged. And here comes the question of the mode of percussion. I do not quite agree with Dr. Lees in thinking that the finger of the observer is the very best method. I think it is the best as a percussor, but I do not think it is as a pleximeter. But you must remember I am a little biassed; I like my little pleximeter best, because I think it gives the most accurate results. I was a pupil of Piorry many years ago, who used to say—I thought then with very good show of reason—that the soft cushion of the finger does not give you as good results as something a little rigid, which shows you the vibrations better. I agree with Dr. Lees in the main; and these remarks are a little outside the practical, perhaps. If you find by the finger the point of extreme pulsation, and then apply your finger outside, you get very fairly the outline of the enlarged heart; that is to say, one finger-breadth away. And that it is enlarged frequently I entirely agree with him; it may be temporarily enlarged, or even in the course of twenty-four hours it may enlarge and then recover, but in other cases it takes several days to recover; in fact, it goes through the phases of an ordinarily enlarged and swollen joint, according to the best of my knowledge and belief. Why does it do that? And let me tell you that that sometimes occurs in a previously diseased heart and sometimes in a sound heart, and the subject of that heart may have no distressful symptoms; it need not have any symptoms of distress and trouble. That being so, why is it so? The heart, according to some members of our profession, and according to some observers, is dilated. Well, it may be so. Why is it dilated? Is it dilated because there is any particular form of myocarditis? I do not think so. I am sorry to confess—and this is a Society for the study of disease in children—that I know very little or even nothing clinically about myocarditis. We read of myocarditis, but I do not know any clinical sign by which I could determine that there was myocarditis. I do not know even whether in these cases there could be any degeneration as a consequence of such myocarditis. I should think not, because the heart resumes its normal size and shape so readily and quickly. What do I know about it? All I know is what I have been told, and by personal observation, the opinion which I recorded many years ago in my Lettsomian Lectures, that the heart is infiltrated under such circumstances, and I demonstrated that it is infiltrated. And Dr. Fisher, who has written an exceedingly good paper on this subject in the 'Lancet,' is of opinion, as Dr. Lees and Dr. Poynton and Dr. Payne are, that there is something which enfeebles the heart, but it is not of the nature of the ordinary form of inflammatory trouble or degenerative trouble; it is an infiltration which cripples the heart. If that is so, let us think to what very important points it leads us. It is that the dilatation of the heart is not an unusual phenomenon; it is a fairly common phenomenon.

If Dr. Lees is right—and I think he is—it occurs almost invariably in any form of rheumatic heart disease; the heart gets dilated and enlarged. But it does so without any particular sign as regards the patient, and by-and-by it gets all right again. Some observers who hail from Germany and tell us that dilatation of the heart is such a fearful thing that it has to be observed by nurses every hour or two with a sphygmograph, and the patient's attention must be constantly directed to it, tell us what is an unreasonable thing, inasmuch as it is accompanied by no special signs, and the patient makes a very good recovery in a considerable number of cases, though not in all. But even suppose there is a heart already diseased, a temporary dilatation that accompanies the rheumatic process, it does not make a great deal of difference unless the disease within the heart increases, and that it very often does from pericarditis. But I do not think we may say that dilatation of the heart, as such, is a thing of the greatest immediate significance and importance; and I may go further, and say that dilatation of the heart, in some cases even of valvular disease, is a very good job. If you have a crippled mitral valve, for example, a regurgitant condition, a condition of mitral insufficiency, it is a very good thing to have some dilatation of the left ventricle. You would not get compensation unless you had. In such a case the left ventricle drives some of the blood back into the left auricle, and does not send so much into the aorta. But if by-and-by, in the process of time, it is to send sufficient for the nutritive needs of the organism it must hold more, and to hold more it must be dilated. Therefore I do not think we can agree that dilatation is always of the grave and severe significance that some people think. I quite agree that in aortic disease painful conditions occur, and that there may be something of distension of a serious kind in that condition; but in the great majority of cases the dilated heart is not of necessity of the significance that some people have made out.

So much for polemics. Now, to come to the other groups, I will just say this. From what I have said already, that the heart of rheumatism is a heart that enlarges, and rheumatism in all forms is a dangerous thing as regards the heart, and produces organic disease, the rheumatic-gout or osteo-arthritic form is not productive of any disease of the heart, but over-sensitiveness of the nerve mechanism of the heart.

The heart of rheumatic pericarditis I have not time to consider now. With regard to the heart of rheumatic endocarditis with ulcerating valvular disease, we all know the extreme significance of that. And the heart of slow and insidious rheumatic endocarditis inducing mitral stenosis I think I should say a little upon, because it has been a subject of very long study with me. The disease known as mitral stenosis is a very subtle one, so subtle that it has been considered, as I have said before, to be a form of congenital anomaly. I do not think it is. I think it is the result sometimes of congenital trouble; that it may occur in intra-uterine life; that it is very subtle and free from symptoms in its origin and progress, and it is only, generally, when the child is seven to twelve years of age that it becomes strongly pronounced and is detected. The smoothness of the funnel-shaped mitral has sometimes suggested that it is a congenital anomaly, but I believe it is not so. The disease is rheumatic, and only rheumatic, because I have seen those cases ultimately, and in the course of time, develop true and undoubted rheumatism, although some observers, especially French, have thought, from the insidious character of it, that it is not rheumatic *ab origine*, but tuberculous. I think there is no such likelihood.

I have been able to only very slightly skim over the subject. I have endeavoured to bring before you what my conception of rheumatism with regard to the heart is. My opinion is not worth very much, and it is only worth what it is if you endorse the opinions which I give, and submit them to that actual study which is the province of this Society. But I do think that we should alter, and try and persuade our professional brethren to alter their conceptions of rheumatism, and to remember that rheumatism is a very subtle and gradual form of disease; that all arthritis is not rheumatic; in fact, there are many other causes of arthritic disease in children as well as in adults that are not rheumatic, and that the heart affections are peculiarly symptomless, very often in their origin and progress, and that the treatment of them must be conducted on rational lines, and that anything of the *ad captandum* ought not to be applied to them. (*Read June the* 21*st*, 1902.)

71. ATRESIA OF THE CONUS PULMONALIS.

By EDMUND CAUTLEY, M.D.

This case illustrates an uncommon form of congenital heart disease, namely, atresia of the conus pulmonalis and patency of the septum ventriculorum, without any malformation of the pulmonary valves or artery. It gives additional support to the theory that many malformations of the heart are associated with, if not actually due to, congenital syphilis. Other notable features are the absence of an extreme degree of cyanosis, and the prolongation of life for a year, in spite of the great obstruction to the passage of blood to the lungs by way of the pulmonary artery. The following are the notes of the case.

Alfred W—, aged 12 months, was admitted into the Belgrave Hospital for Children on November the 28th, and died on December the 9th, 1901. He was the first child, was born at full time, and was breast-fed up to the date of admission. His mother appeared healthy, had had no miscarriages, and gave no history of symptoms indicative of syphilis. The child first came under observation as an out-patient on September the 3rd, and was brought to the hospital fairly regularly up to the day of admission. From the age of one month he had been subject to occasional "fits," to coughing which was often paroxysmal, and to attacks of blueness. *On examination*, there were noted coughing, frequent respirations, slight beading of the ribs, signs of congenital syphilis, hypertrophy and dilatation of the heart, and a systolic cardiac murmur. The main point of interest recorded in his out-patient notes was the increased frequency of breathing, although there was nothing pulmonary found to account for it beyond an occasional *râle*. The dyspnœa appeared to be essentially cardiac. The duskiness was only slight, and attracted little attention. *On admission*, he weighed 11 lbs. The complexion was somewhat earthy, and the mucous membranes and extremities were a little dusky. There was no "clubbing." The signs of congenital syphilis consisted of purulent nasal catarrh, extensive linear scarring of the lips, a general erythemato-squamous rash on the trunk and limbs, and superficial serpiginous ulceration about the anus. Phimosis, balanitis, and considerable œdema of the scrotum were also noted. Nutrition was distinctly poor. The heart was much enlarged, being hypertrophied and dilated. Pulsation could be felt two inches to the right of the sternum. Its action was abnormally frequent. A systolic murmur could be heard all over that portion of the heart which was situated to the left of the middle line of the sternum. It was soft, and was best marked over the pulmonary area. It was heard over the aortic region, but was not conducted into the vessels in the neck, and it could not be heard over the back. There was no thrill. In view of the malformations found post mortem, it is rather remarkable that the murmur was not louder and more widely distributed. There was no evidence of lung mischief. The liver was enlarged. The spleen could not be felt.

December the 2nd.—To-day the child had a bad attack of dyspnœa and became very blue. The attack lasted for a few minutes, and both pulse and respiration were very frequent.

5th.—He has had three attacks of dyspnœa since the last note, but none of them were very bad. There is a loss of 3 oz. in weight in a week. He takes his food well, but does not thoroughly digest it. The murmur is a little louder at the apex, and the heart is rather more dilated. No adventitious sounds can be heard in the lungs.

7th.—Another very bad attack of dyspnœa to-day.

9th.—He has had more attacks of dyspnœa, and he died to-day from cardiac failure during an attack.

The pulse-rate varied from 144 to 162 beats per minute, and the respiration-rate from 44 to 64 per minute. During the attacks of dyspnœa both the pulse-rate and the breathing became still more accelerated. The temperature chart showed some irregular pyrexia, which might have been due either to insufficient aëration of the blood supplying the thermotaxic centre or to gastro-enteric irritation.

Post-mortem examination.—The lungs were a little engorged. The heart was very much enlarged and globular in shape. The right ventricle was as large as the left, both as regards the cavity and the thickness of the walls. The interventricular septum was represented by a well-developed muscular partition in its lower two-thirds, ending at its upper edge in a thick, smooth, rounded border. The orifice thus left between the two ventricles would admit the little finger. The aorta was large, dilated, and arose from both ventricles, communicating equally freely with each. Its valves were normal, but hardly seemed large enough to close the orifice during diastole. In the wall of the right ventricle, high up, in the part which normally forms the conus pulmonalis, was an orifice, which would admit a No. 1 catheter, leading into a small thin-walled pulmonary artery. The pulmonary valves were more delicate and smaller than normal, and very much more delicate than the aortic valves. The ductus arteriosus was impervious, but a fine probe could be passed into it from either artery almost to the middle of it, although there was no through passage. Its condition suggested comparatively recent obliteration. There was a valvular orifice in the foramen ovale. No other malformations were found.

It is often comparatively easy to diagnose congenital heart disease, and superlatively difficult to ascertain the exact lesion. Let us see if this case affords any useful indications for diagnosis. It was perfectly clear that there was cardiac malformation. The presence of some degree of cyanosis, a very frequent pulse and respiration rate, and a systolic murmur were conclusive. The character and the localisation of the murmur indicated some form of pulmonary obstruction. In view of the malformations found after death I am of opinion that the murmur was due to the obstruction to the flow of blood through the narrowed conus; that on account of the excessive narrowness very little blood could get through and only comparatively slowly, hence the murmur was not loud, and there was no thrill. I consider that the patent septum was not responsible for the murmur. Its edges were beautifully smooth and rounded, and the blood could flow with perfect ease from either ventricle into the aorta. It is more than probable that a murmur due to this cause would have been readily conducted into the great vessels, and would have been heard behind. If this conclusion be accurate it follows that patency of the septum ventriculorum can exist without the production of a cardiac murmur. It was astonishing that, with such an extreme degree of atresia, the lividity was not more marked. Possibly this may have been partly due to delayed closure of the ductus arteriosus, and partly to the blood being carried to the lungs by the bronchial arteries. Unfortunately I was not present at the post-mortem examination, and the bronchial arteries were not examined. The slight increase in the loudness of the murmur at the apex was probably due to dilatation of the ventricles.

If we look at the case from a developmental point of view we must bear in mind that the heart is nearly as well formed at the beginning of the second month of fœtal life as it is a few days before birth. Before the end of the second month all the great antenatal developmental processes are completed, and subsequently the heart merely grows in size and weight. It therefore follows that the malformation was due to causes acting during the first few weeks of fœtal life. For some reason the conus pulmonalis was not properly formed, and, in consequence of the obstruction to the flow of blood, the septum ventriculorum was not completed. Such a malformation could hardly have been due to a fœtal endocarditis, and is almost certainly due to an error of development. Alcoholism, syphilis, and tuberculosis in the mother are the most common causes of developmental errors, and, in the present instance, there was conclusive evidence of congenital syphilis.

The view that congenital heart disease is generally due to fœtal endocarditis is much too prevalent. It is barely possible that endocarditis can cause malformations by the arrest of formative processes during the first two months of life. Some of the cases examined post mortem show that the damage is due to errors of development. Thus, of the last forty instances of congenital heart disease which have come under my notice, eight have been examined post mortem with the following results.

1. Jessie Y—, aged 10 weeks. The pulmonary artery arose from the aorta, the vena cava inferior opened into the left ventricle, the septum ventriculorum was patent, and the foramen ovale was fenestrated. The mother had had rheumatic fever, and one of her children had a congenital sacral cyst. Five of her children had died under two years of age. There was no history of syphilis.

2. Edward W—, aged 4 months. A trilocular heart, there being only one ventricle and a patent foramen ovale. There was no history of rheumatism or syphilis.*

3. John M—, aged 10 months. The foramen ovale was patent. The baby had been blue since birth, but there was no cardiac murmur. There was no history of rheumatism or syphilis.

4. Rosie L—, aged 15 months. The ductus arteriosus was patent, and one cusp of the aortic valve was thickened. There was no cyanosis during life. There was no history of rheumatism or syphilis.

5. Florence S—, aged 22 months. The mitral valve was represented by thickened, shrunken, fibrous masses of horseshoe-shape. The physical signs during life were those of advanced mitral regurgitation. The mother had mitral disease.

6. Florrie N—, aged 15 years. Pulmonary regurgitation secondary to pulmonary stenosis.†

* 'Reports of The Society for the Study of Disease in Children,' vol. i, p. 132.

† 'Reports of The Society for the Study of Disease in Children,' vol. ii, pp. 44—50.

7. Thomas B—, aged 14 years. Pure aortic stenosis.*

The eighth case is the one here recorded, and, like Cases 1 and 2, is undoubtedly due to developmental error. Cases 4 to 7 were probably due to fœtal endocarditis. The cause in the third case is doubtful. Thus there is certainly a preponderance of post-mortem evidence in favour of fœtal endocarditis, but some cases are undoubtedly due to errors of development.

In one of the thirty-two other cases there was very strong proof of syphilis acting as the exciting factor, for the mother was delivered of a still-born child, previous to the birth of the patient, while she was actually under treatment for syphilis. In several instances the family history was strongly suggestive of syphilitic infection. On the other hand, it is by no means uncommon to find a history of rheumatism in the family, and even in the mother herself.

We shall, therefore, be justified in dividing cases of congenital heart disease into two groups:

(*a*) Cases due to developmental errors during the first two months of intra-uterine life; the most probable cause of such defects being inherited syphilis.

(*b*) Cases due to fœtal endocarditis.

Under the second group would be included all those cases of valvular lesions leading to adhesion, contraction, or destruction of valves, and many instances of delayed closure of the foramen ovale and ductus arteriosus. It must also be borne in mind that the two factors may be co-existent in any one case, for endocarditis is more likely to attack a malformed than a sound heart. Moreover structural defects, malformations, and endocarditis may be due to a common cause which has begun to act in the embryonic stage of fœtal life.

Discussion.

Dr. Theodore Fisher (Bristol) said he would respond to the Chairman's invitation by making a few brief remarks on Dr. Sansom's paper. As Dr. Sansom would no doubt agree, the most striking feature of heart disease in children was pericarditis. Pericarditis occurring before twelve years of age almost invariably limited the patient's life to twenty years. As his own few statistics and the more extensive ones of Dr. Sequeira showed, the subjects of that condition in childhood generally died at about the age of puberty—between thirteen and fifteen years of age; very few reached the age of twenty years. Practically no cases lived to twenty-five years. Dilatation of the heart in rheumatism was an interesting point, and he thought that, in the main, what Dr. Sansom had said was true, that many of the slight dilatations which occurred in the course of rheumatism were probably not of a serious nature; and many of those cases which were thought to have a mitral lesion because a mitral systolic murmur was heard at the bedside, if they were traced would be found later to have lost that murmur. He hoped at some future time to investigate such cases; to take a number of patients who developed a mitral systolic murmur, and see what happened to them. Dr. Caton (of Liverpool), who kept such cases in bed longer than most physicians, noticed that the murmur often disappeared. Dilatation, slight in amount, occurring in rheumatism, was not serious. Going back to pericarditis, and referring briefly to myocarditis, to which Dr. Sansom alluded, he (Dr. Fisher) thought the presence or absence of myocarditis in rheumatism depended on the severity of the poisoning of the heart. The question was a large one; but he thought the association of myocarditis with pericarditis showed that the heart was poisoned as a whole. In those cases of intense poisoning, whether the heart muscle showed any obvious lesions in the post-mortem room or not, the muscle was weakened in some way during the period of growth, and so seriously that it gave out early in life. In many cases, even of pericarditis, which came to the post-mortem room one could not find sufficient lesions of the heart muscle to account for death; but there were cases in which such lesions were very well-marked indeed, even where pericarditis was slight in degree. He had one case recently in which the pericarditis was limited to the auricles, and there was a patch of old adhesions in front of the ventricles about an inch across. The rest of the pericardium was healthy, but the myocarditis was intense, was visible to the naked eye, and under the microscope every muscle-fibre proved to be affected by the fatty degeneration. With regard to congenital disease of the heart, he agreed with Dr. Cautley's classification. Cases having a patent foramen ovale could hardly be put in the same class as other congenital malformations, unless it were very marked in degree. It was a point not generally recognised by pathologists, although anatomists might know of it, that a slight patency of the foramen ovale could be found in about one-fourth of the adult cases, if it were looked for. He was struck with this fact when he was distending hearts with water, as mentioned in a recent article in the 'Lancet.' In one-fourth of those cases both sides of the heart could be distended by injecting fluid into one or other side, on account of the patency of the foramen ovale.

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* 'Med. Soc. Trans.,' vol. xxiii, pp. 324–5.

72. SOME REMARKS ON THE RESULTS OF TENDON-TRANSPLANTATION IN TWENTY-FIVE CASES OF INFANTILE PARALYSIS OF THE LOWER EXTREMITY.

By W. P. MONTGOMERY, B.S.

Though suggested by Nicoladoni as long ago as the year 1882, it is only within the last few years that serious attention has been directed to the operation of tendon-transplantation. Revived by Drabnik in 1894, and practised by Vulpius, Eve, Lange, Robert Jones, Tubby, and others, the principle has been shown to be one that is sound, and the results that have been obtained have been increasingly good.

In the present paper I shall confine myself to a brief summary of the points which have presented themselves to me in a series of twenty-five cases.

Of the classical methods of operation there are four, which are as follows:

(1) The division of the tendon of a healthy muscle, and its implantation into the tendon of one that is paralysed.

(2) The division of the tendon of a paralysed muscle, and its attachment to an acting undivided muscle (the "passive" method of Hoffa).

(3) The transference of a slip from an active muscle tendon to one that is paralysed (the "active" method of Hoffa).

(4) The attachment of an active muscle tendon, or of a slip from it, to the periosteum at a new site.

Each of these methods, and in some cases a combination of more than one, has been brought into use in my cases.

The cases fall naturally into five groups:

(1) Those in which a slip from the tendo Achillis was used as a graft to other muscles, *viz.* to the extensor communis, two cases ; to the peronei, three cases ; to the tibialis posticus, three cases ; and to the tibialis anticus, one case.

(2) Those in which the acting extensor longus hallucis tendon was divided and transplanted to the tibialis posticus tendon, one case ; to the extensor communis tendon, two cases ; to the tibialis anticus tendon, two cases.

These were the only instances in which the so-called old method was employed. Generally speaking, it is not considered advisable to sacrifice, or risk the sacrifice of, a healthy acting muscle. But as the great toe has a subsidiary extensor in a slip from the extensor brevis digitorum muscle, we may safely make the long extensor an exception to the rule.

(3) A slip from the tibialis anticus tendon was attached to the extensor communis tendon in three cases ; to the tibialis posticus tendon in two cases ; to the peroneus longus tendon in one case.

(4) Cases in which a slip from an acting muscle tendon was transferred to a new site on the periosteum. In two cases the Achilles tendons and in two the tibialis anticus tendons were employed. In all the slips were attached to the periosteum over the cuboid bones. These were cases of paralysis of the peroneal type.

(5) Cases in which the transplantation was associated with the removal of some portion of bone in order to correct deformity. This group includes four cases, in all of which the partial or complete removal of the astragalus was performed.

In the great majority of instances the methods adopted were applied for the correction of deformity caused by the paralysis of one or more of the dorsiflexors of the ankle-joint. In all the cases the paralysis was permanent, and had lasted for more than a year. Two cases were operated upon over four years ago, but the great majority have only been done from six to twelve months. It would, therefore, not be right to dogmatise on the ultimate results. Still, I may justifiably come to some conclusions, which I would summarise under the following sections.

Conclusions.—(1) The progress of cases in which the gastrocnemius and soleus muscles were adapted as dorsiflexors of the ankle-joint was very slow. Still there is no doubt that the work of re-education goes on surely, and the cases improve steadily. It is here that the graft-tendon acts also as a mechanical support to the foot and ankle in the way pointed out by Eve. In cases in which the transplanted and paralysed muscles were more nearly allied in their duties the improvement was much more rapid, and the earlier results apparently much more satisfactory.

(2) The longer the length of the tendon of a paralysed muscle to which the transplanted muscle was applied, the less satisfactory was the early result, and the slower the improvement. There is no doubt that this factor of the stretching of the paralysed tendon or its muscle is a most important feature. It was to obviate this difficulty that Lange suggested that the slip from the transplanted muscle should be fixed to the periosteum at a suitable site. This, of course, cannot always be done without so lengthening the transplanted tendon as to considerably weaken it. In the four cases in which this method was used the improvement was certainly both quicker and more definite.

(3) The results of the passive method, division of the paralysed tendon and its suture to the intact acting one, were much inferior to those of the active method in which a slip was taken from the normal tendon. It is better too, in my experience, to divide the paralysed tendon and fix the graft to its cut end, rather than to merely button-hole it and attach the slip to the tendon in its course. In the cases in which this latter method was employed the improvement was very slow from slackening of the paralysed muscle. Whenever at all possible the other alternative should be chosen. One must be convinced that no spontaneous improvement can take place before cutting the tendon across. If there be any doubt, the graft should be applied to the tendon in its course.

(4) There is no doubt that at the time of the transplantation all deformity must be corrected. In four cases this required the excision of more or less of the astragalus, and they all did well. In several others tenotomy, or tendon lengthening or shortening were performed. This lengthening or shortening gives a much better result than simple tenotomy alone, and this is especially true in the case of the Achilles tendon in instances of talipes equinus and talipes equino-varus.

(5) As regards after-treatment, the ideal method is to begin massage and passive movement in three weeks' time and gentle active movements a week later. With patients of the hospital-class of course this cannot be done, and with them I think it best to fix the foot in plaster for five or six weeks, and then allow both active and passive exercises. I have been unable to convince myself that either faradism or galvanism contributed to the improvement of any case. But these agents were usually very intermittently applied. In certain cases where stretching of the tendon might be anticipated, I used an artificial muscle for the first few weeks in order to lessen the strain. Generally, no apparatus of any kind is either advisable or necessary. I used silk as the suture medium in all my cases, and had no instance of a stitch-abscess or other trouble. Absolute asepsis is necessary for the success of the procedure. (*Read June the* 21*st*, 1902.)

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73. THE SURGICAL TREATMENT OF A CASE OF INFANTILE PARALYSIS OF THE LEG.

By C. N. GWYNNE, M.D.

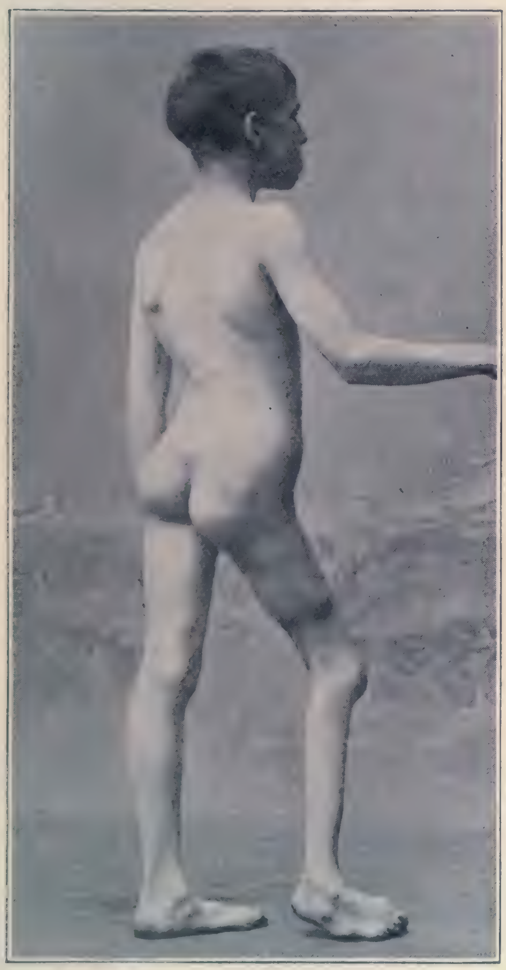
John B—, when aged 6 months, developed an attack of infantile paralysis, which affected the right leg from the hip downwards. The family history was good. He was one of a family of eight children, all of whom except himself, and an eldest sister who suffered from congenital dislocation of one hip, were healthy. His mother died at the menopause of heart disease with an enlarged liver and dropsy. The boy John grew up to be a fine healthy lad. With the aid of a leather splint laced up the front and strengthened by side irons extending from the boot to the upper part of the thigh, he was able to get about with a waddling gait, supported by a single crutch, pushing his leg along by the action of the upper thigh muscles. The limb did not grow *pari passu* with its fellow either in length or thickness, and at the time he came up for surgical treatment it was two inches short at least. As is usual in such cases its muscles were atrophied, and its temperature below normal.

At the time he presented himself for operation the condition was as follows:—The limb was rather more than two inches shorter than its fellow, and the extensor muscles of the leg were paralysed, so that he could not even to a slight degree extend his leg. The muscles around the hip-joint acted partially, the psoas and iliacus muscles retained some power, and he could to a slight degree raise the thigh on the abdomen. The action of the flexors of the knee-joint was fairly good,—too good, in fact, for by their constant action without opposition they gradually produced a dislocation backwards of the leg, which was ultimately drawn up behind the condyles of the femur.

The leg from the knee downwards was like a flail, and it was pulled outwards by the external thigh muscles, forming a bad genu-valgum.

Reviewing the condition of the ankle and the foot, we were brought face to face with very complex abnormalities. Partly owing to the displacement of the leg backwards at the knee and partly to the action of the relatively large or rather prominent external condyle, which contributed to an outward rotation of the tibia, the ankle-joint faced almost directly outwards, and the fibula was brought into a position directly behind the tibia. Here the early conditions produced a talipes valgus; the heel was turned outwards, and the impact when walking was on the inner border of the foot (*vide* Fig. 24).

FIG. 24.



In the course of time this position became at first modified and subsequently was completely altered, so that what was originally a talipes valgus became transformed into a talipes equino-varus. The course of events that led to this was, I take it, the following:—As the tibia rotated outwards the tibialis anticus muscle, aided by the extensor longus muscle, which retained some of their energy, drew the foot inwards, producing a subluxation of the astragalo-tarsal joint, and at the same time the action of the muscles that operated through the tendo Achillis pulled inwards, and subsequently upwards, the lower plane of the os calcis. The foot thus became sickle-shaped, with the curve facing inwards, and as the changes thus initiated gradually progressed the plantar surface of the foot finally faced inwards, and the impact was transferred to the outer edge of the foot, where in time extensive callosities formed.

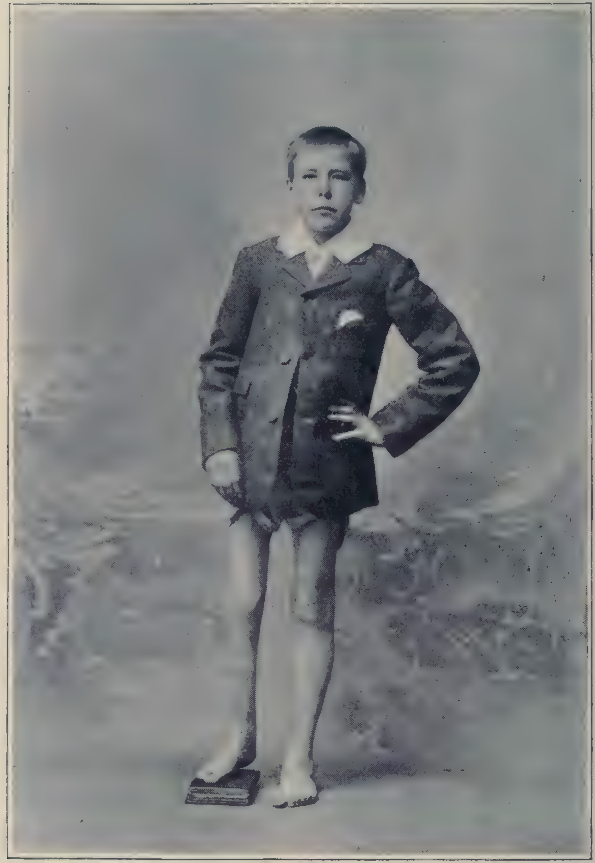
On examining the foot at this period the head of the astragalus was prominent and faced outwards, and the articular cavity of the scaphoid had left it, and articulated with the inner surface of the head and neck of the astragalus. The articular relationships of the astragalus and os calcis were also disturbed. The latter bone only partially followed the astragalus in facing outwards, while at the same time the inner tuberosity was drawn upwards as previously mentioned, the outer tuberosity alone being in contact with the ground. All the muscles of the foot were almost quite paralysed excepting the tibialis anticus, the extensor communis, and the flexors of the toes. These muscles retained only a very feeble power of action. In the treatment of the case everything that could be done by the aid of leg instruments, by massage, and by electricity, etc., was done, but the condition remained very unsatisfactory; the spine was becoming curved laterally, and the foot was constantly suffering from pressure sores. On consideration I came to the conclusion that arthrodesis offered a fair chance of success, and if it failed an amputation above the knee could be resorted to.

On October the 10th of last year (1901) I resected the knee-joint, Dr. Wilks, my colleague, assisting me. I removed barely enough of the joint to be clear of the articular surfaces, and after some trouble in reducing the backward luxation I succeeded at length in getting the surfaces of the bones in good apposition. I did not leave the patella. Two days afterwards I put the leg into a plaster splint, and did not remove it for nine weeks. The appearance of the leg on the removal of the splint was good. There was no extra shortening, as the removal of portions of bone was fully compensated for by the reduction of the backward luxation.

On February the 2nd of this year I operated on the ankle-joint. What I proposed to do was, firstly, to remove the articular surfaces of the tibia and astragalus with as little loss of bone as possible, and then to rotate inwards the astragalus, so as to bring it back as far as possible to its normal relationship with the bones of the tarsus. Secondly, to remove the articular surface of the scaphoid, so as to favour bony union between it and the astragalus. Thirdly, to rectify the sickle shape of the foot by section of its ligaments and deep fasciæ.

The following were the steps of the operation :—An incision was made on the outer side of the ankle-joint, and subsequently a very limited one on its inner side. Care was taken not to wound any vessel of importance, as the vitality of the limb was low. A dissection was made close to the bone which united the two incisions, and with a fine saw the upper articular surface of the astragalus was removed. Next the articular surface of the tibia was removed, and with it the internal malleolus, which, by its anterior position, would have prevented the inward rotation of the astragalus. The internal lateral ligament was divided, and with a gouge the articular surface of the scaphoid was removed. The opposing cartilages of the astragalus and os calcis were also gouged away, and the deep fascia on the inner aspect of the foot being divided, the foot was forced into a correct position. There was no bleeding to speak of, and after the first dressing the foot was put up in plaster, and retained in it for a couple of months. I took it out of the plaster about the end of April, but reapplied it, being content with a very light splint. At first he was very nervous about putting his full weight upon the leg, but he is gradually getting over that feeling, and when I last saw him he could walk about the room without a stick with increasing confidence (*vide* Fig. 25). (*Read June the* 21*st*, 1902.)

Fig. 25.



Discussion.

Mr. Clement Lucas, in response to the Chairman, said he would have preferred to hear the remarks of Manchester members. Having seen Mr. Montgomery's and Mr. Gwynne's cases, he could say they afforded excellent examples of what could be done now for cases of infantile paralysis, which a few years ago were considered to be almost hopeless. Indeed, they were cases in which one always, after dividing tendons to correct the deformity, warned the parents that in all probability the deformity would shortly recur. If that were not done, the patient's friends were very discontented with the results of the tenotomies, which were then the only means available of correcting the deformities. Treating such cases by displacing the tendons of acting muscles, and joining them into positions where they would correct the deformity and assist in the movements, was a very great advance. He had looked over Mr. Montgomery's cases with very great pleasure, and there was very little he could add to what had been said. He had himself done more in the way of excising the bones in the extreme cases of deformity than had been done in some of the present series. In one of Mr. Montgomery's cases he thought a little more of the head of the astragalus might have been removed, so as to get down the hump. The way in which one could bring an acting tendon into a new position, so as to support an arch or alter a deformity, was one of the greatest recent advances in the treatment of such cases.

Mr. Walter Edmunds said he was very much interested in seeing Mr. Montgomery's cases, which he thought were very successful. He refrained from saying more, as he did not wish to delay the meeting.

Mr. J. Collier (Manchester, introduced) said he had performed tendon-grafting on very few cases. He agreed with Mr. Montgomery that the best way was to graft tendon on to periosteum if that could be done and the tendon were long enough. He had greater experience in the way of arthrodesis in cases where the limb was flail-like. A child whose ankle-joint he had fixed could now be seen walking about. The child brought that leg forward merely by the increased flexibility of the limb. In doing an arthrodesis, in a case where the ankle-joint was flail-like, he had found that it was still more difficult to get that joint fixed than it was to fix a knee-joint.

74. A CASE OF DISPLACEMENT OF BOTH TESTES *IN PERINÆO*, WITH AN INGUINO-PERINÆAL HERNIA ON THE RIGHT SIDE.

By R. CLEMENT LUCAS, B.S.

H. W—, aged 3 years, was admitted into a cot in Lazarus Ward, Guy's Hospital, on May the 2nd, 1902, on account of a swelling in his right groin and perinæum.

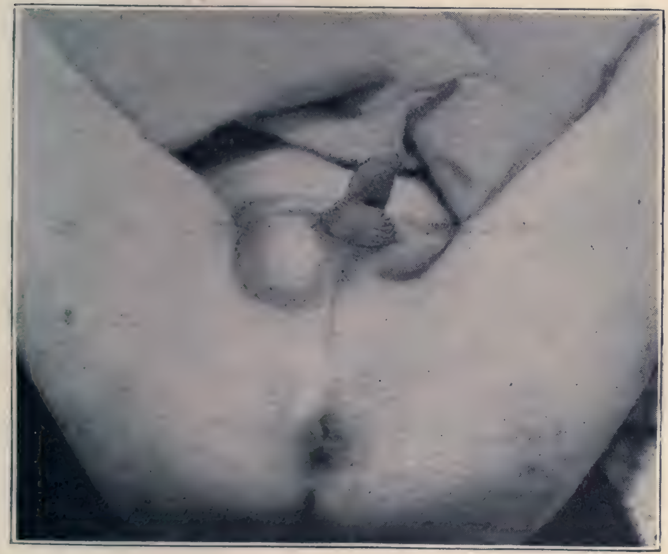
The boy was a fairly healthy son of somewhat unobservant parents. They had apparently noticed nothing amiss with him during his infancy, and, except for slight troubles when teething, his general health had been uniformly good.

One day the boy was trying to climb on a chair when he suddenly began to cry, and put his hand on his right groin. His mother looked and found a swelling there. She took him to a doctor, who said he had a rupture and a misplaced testis, and advised her to take him to a hospital.

When admitted he was found to be suffering from a hernia, which extended from the inguinal canal, past the outer side of the scrotum, into the perinæum behind this pouch. It was somewhat tender, but reducible. At the back of this swelling the right testis could be felt fixed in the perinæum. On the left side there was no hernia, but a swelling was evident just behind the left half of the scrotum, and this, when grasped, could be felt to be the left testis. The penis was well developed, but the scrotum was small, puckered, and empty (*vide* Fig. 26). Physical examination thus clearly proved that the case was one of double *testis perinæalis*, which had been overlooked until a hernia descending into a congenital sac on the right side drew attention to the congenital misplacement.

Operation on the right side.—On May the 14th A.C.E. mixture was administered, and an incision was made over the external abdominal ring. The cord was exposed and the testis drawn up. It was found attached by a strong gubernaculum to the tissues of the perinæum, which required division before the organ could be raised. The congenital sac of peritoneum was then isolated, and excised from the internal abdominal ring to the top of the testis. The lower portion was sewn up by a fine silk suture to form a tunica vaginalis, whilst the upper end was transfixed, ligatured, and tied to the abdominal muscles. The right side of the scrotum was then invaginated, and two small silk sutures were used to fix the testis to the bottom of the scrotum. Next the inguinal canal and external abdominal ring were narrowed and strengthened by cross silk sutures cut short and buried, and finally the skin was sewn up with a horsehair continuous suture, and the wound sealed with collodion and iodoform. No drainage was employed, and no incision was made into the perinæum. The wound healed primarily, and the child had a normal

Fig. 26.



temperature for six days, when it suddenly ran up, and later he developed the coryza and rash of measles, and had to be sent to an isolation ward.

The measles ran a normal course, and in no way interfered with the proper union of the parts severed in the operation. He was discharged from the hospital on June the 12th, convalescent from measles, and with the wound soundly healed.

On June the 19th he was brought again to the hospital with the object of having the left testis implanted in the scrotum. On examining him it was found that no further operation was necessary; as the result, apparently, of the double spica bandage carried from thigh to thigh and across the perinæum to retain the dressings in place after the operation on the right testis, the left was now found to be in its proper position in the left side of the scrotum. The scrotum had undergone development, and the testis showed no tendency to escape again from the purse.

(*Read June the* 21*st*, 1902.)

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75. FLEETING AMAUROSIS IN INFANTS PRESENTING SYMPTOMS OF MENINGITIS.

By SYDNEY STEPHENSON, C.M.

That infants suffering from meningitis often lose their sight is, of course, a fact known to every student, and it is duly set forth in every text-book of medicine. Indeed, the examination of the eyes with the ophthalmoscope is nowadays rightly regarded as an essential item in the physical investigation of every case where meningitis is suspected. In many of the patients examination reveals a papillitis, which may be followed at a later stage by atrophy of the optic disc, and presumably also of the optic nerve.

There exists, however, a type of case which is less common, where during the course of a cerebral attack sight appears to be altogether lost without ophthalmoscopic signs of either papillitis or atrophy, and the particular interest of such cases lies in the fact that the children often recover both sight and health at a later period. My inquiries lead me to believe that this "fleeting amaurosis" is not so generally recognised by physicians as it might be—an omission the more curious seeing that its essential features have been worked out by several observers, notably by Mr. Edward Nettleship* and by Dr. William Gay,† who have published excellent descriptive papers upon the subject.

In my own experience of these cases the patients are usually below two years of age. There is a history of recent illness, marked by retraction of the head, fits, twitchings, drowsiness, stupor, rigidity, vomiting, bulging of the anterior fontanelle, headache, and perhaps other symptoms suggestive of an inflammation of the meninges of the brain. The temperature may be of distinctly febrile type. Obstinate constipation is sometimes present, although that is by no means the rule. At some period during the illness (which, as a rule, runs a chronic course) the mother finds that her baby ceases to take notice of things and to recognise familiar objects. The loss of sight usually appears to be complete, and, as far as can be made out, comes on almost suddenly. At this stage the pupils, in my experience, are larger than normal, and possess little if any direct response to light; but beyond that I have never succeeded in obtaining any objective evidence of organic changes in the eyes. In the further course, cerebral symptoms pass away, and the child slowly regains its health. Sight gradually returns, at first for large and bright objects, but later for things even so minute as pins lying upon the floor of a room.

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* Nettleship, 'Trans. Ophthal. Society,' vol. iv (1884); 'Roy. Lond. Ophthal. Hospital Reports,' vol. xi (1887), p. 353.

† Gay, 'Roy. Lond. Ophthal. Hosp. Reports,' vol. xiii (1893), p. 404.

The ophthalmoscopic appearances, as might be expected, are then negative. The pupils react well to light.

That a simple basal meningitis existed in most of the cases was the opinion held both by myself and by the physicians in more direct charge of the patients. Such, in brief, are the essential features of what Dr. Gay has proposed to call the "acute cerebral amaurosis of infancy," than which it would, I venture to think, be difficult to find a better descriptive name.

It is impossible to doubt that such cases form a definite group, the existence of which can scarcely be too widely known. I have notes of six such cases, and I will detail briefly four of them that I have been enabled to follow up more closely than the others. In doing this I should wish to thank Dr. George Carpenter and Dr. J. H. Sequeira, under whose care some of my patients originally were, for permission to publish the cases.

Case 1.—Henry K—, aged 7 weeks, was brought to me at the Evelina Hospital on July the 26th, 1901, with the statement that he had lately lost his sight. The child, who was breast-fed, had suffered from vomiting for three weeks and from diarrhœa for a few days. His head was markedly retracted, and this was said to have come on two or three days after the sickness began. The pupils were equal and reacted to light; there was no squint. The optic discs and retinal vessels showed no changes, but the baby certainly appeared to be blind, since it took no notice of a bright light. A maternal aunt had died of phthisis.

August the 2nd.—The baby had several "convulsion fits" soon after leaving the hospital a week ago, and these have been repeated in the interval. He still vomits.

9th.—Although the head is still retracted, the child seems better in himself, and the vomiting has almost stopped. He notices nothing, but the fundi oculorum still show no ophthalmoscopical changes.

23rd.—The sickness has almost stopped, and the child is putting on flesh. The neck is no longer stiff. The pupils are equal, of medium size, but with little reaction to light. The patient recognises his mother when she comes into the room.

September the 20th.—The child now "coos" when his mother comes into the room, and notices things generally. His sight is said to improve daily. The pupils are equal, and act better to light. The fundi oculorum are as at the first observation. His general health appears to be now satisfactory.

Case 2.—Nellie A—, born on April the 19th, 1900, was sent to me by Dr. George Carpenter at the age of about one year (March the 29th, 1901). The history given by the mother was that until Christmas, 1900, the baby was "a pretty little child, quite good and sharp," but that she then developed "congestion of the lungs and bronchitis," and that this was followed by "fits" (seventeen in twenty-four hours). During these fits the sight failed. A private doctor is said to have diagnosed "meningitis" and "water on the brain." The baby did not cry, but lay unconscious for about six weeks. The mother thinks that the baby was blind until the last two or three days, but says that she now appears to notice a little. A brother, a sister, and two cousins of the patient's father have been, or now are, in asylums for the insane.

Upon examination, the pupils were found to be of medium size with little direct action to light. The baby did not follow or notice a light, and seemed to be blind, but the optic disc, yellow spots, and retinal vessels showed no morbid changes.

May the 24th.—Three days ago—that is fifty-three days after I first examined the child—she was noticed to turn her eyes up when the mother approached the cot without noise, and since then she is said to have looked about her in a dazed and nervous sort of way. She has not yet recognised anybody. The mother herself is now convinced that the baby sees a little. The pupils are semi-dilated; the fundi oculorum are normal.

31st.—The mother is satisfied that the baby's sight is slowly returning. The pupils remain of medium size and motionless.

July the 12th.—The child is stated to lose consciousness, and to lie for two hours at a time prostrate. This has happened five times during the present month. She seems to have pain in her head, and "tears, and scratches, and screams." She does not sleep well. She vomited whilst in the out-patient room. She grasps at a gold watch held before her.

August the 9th.—Since her last visit she has suffered from diarrhœa and vomiting. She now obviously takes notice of things, plays with her toys, and recognises her friends. The mother thinks, however, that the sight is still peculiar, as it seems to "fade away" when the baby is poorly. The pupils are equal (2·5 mm.), and react, although not very vigorously, to light. The child notices things at once, and follows them with her hands. She is now sixteen months old. Under chloroform the fundi oculorum were examined thoroughly, but with negative results.

October the 4th.—The infant is now fat, and well in general health. She is restless, always turning and twisting about upon her mother's lap. She often holds her head with her hands. She is stated never to be asleep, and always to be "grizzling like a pigeon cooing." She can sit up, but cannot crawl. She knows her mother and everybody around her, and plays with her toys as a child should do. She is not deaf. The pupils react to light. The fundi oculorum (examined under chloroform) show no changes, the discs are of good colour, the vessels normal, and there are no changes in the yellow-spot regions. Dr. George Carpenter, who saw the case a few days later, thought that she was mentally afflicted.

The next case is of unusual interest, owing to the fact that, although the blindness disappeared, a paresis of one of the extrinsic muscles of the eyeball remained as permanent evidence of the meningitic attack.

Case 3.—Sylvia C—, aged 8 years, was brought to me on July the 19th, 1898, because she squinted, looked sideways at her book, and was said to go downstairs "like an old woman." According to the clear history given, the child when about six months old had a bout of vomiting, which was put down to "teething." At nine months of age, however, sickness recurred, and was followed by muscular twitching, retraction of the head, and failure of sight. For eleven days her temperature ranged between 97° F. and 101° F., but was above normal nearly the whole time. Before that the temperature had reached 103·4° F. on two occasions. There was defective vision, conjugate deviation downwards, and rolling of the head, and it was noted that the dilated pupils reacted feebly to light. The ailment was diagnosed as *tuberculous meningitis* by the physicians in charge, and it was thought by them that the patient could not possibly get well. But, to everybody's astonishment, improvement set in and sight returned. There was, it appears, a relapse, and sight again failed, so that the child is said once more to have become perfectly blind. After the lapse of three or four months the baby was observed to pick bits off her mother's dress, and soon after that is stated to have seen as well as anybody else. There was no phthisis on either side of the family. The patient was the ninth of ten living and healthy children.

On examination, I found there was a paresis of the internal rectus of the left eye, causing an outward squint of 35°—40°. With spectacles the sight of the right eye was normal, and that of the left one-third normal. The pupils were equal and active. The fundi oculorum showed no changes, the optic discs were of good colour, and the retinal vessels normal.

Case 4.—Martha F—, 15 months old, was first seen on April the 22nd, 1901. The child was taken ill three weeks previously, when she had a "fit" and was drowsy for a couple of days. She vomited at the time of the "fit," but has not done so since. The bowels have been loose and the motions offensive. The paternal grandmother died of phthisis. There are five other children in the family, all healthy. *Upon admission,* the child was drowsy, with slight retraction of the head. The anterior fontanelle was much bulged; the reflexes exaggerated. There was neither squint nor optic papillitis. The temperature was 100° F. She weighed 16¾ lbs. A diagnosis of posterior basic meningitis was made by Dr. J. H. Sequeira, under whose care the child was admitted into the North-Eastern Hospital for Children.

April the 29th.—The child is sick three or four times a day. The drowsy condition persists; the anterior fontanelle is still much bulged. The temperature ranges from 97° F. to 100·8° F.; the pulse varies from 76 to 152 beats a minute; and the respirations from 28 to 36 times a minute. The bowels tend to be costive.

May the 3rd.—Vomiting has ceased. The temperature has risen to 101° F., the pulse-rate to 140 per minute. and the respirations to 36 per minute.

10th.—Sickness recurred on May the 7th, and the child has vomited once a day since that date. During the last five days the anterior fontanelle has gradually become less prominent, and is now, if anything, depressed. The temperature has not risen above 99·6° F. since the last note.

17th.—To-day *the child recognised* and talked to her parents when they visited the hospital.

24th.—The temperature has risen once only (101° F.) since the last note. The child is more fretful, and the anterior fontanelle is more prominent. The reflexes are brisk, but the knee-jerks are obtained with difficulty.

30th.—The pupils are dilated and do not respond to light, and the patient is apparently unable to see.

June the 6th.—The child takes no notice of anything, and appears to be quite blind. The pupils do not react to light; the fundi oculorum show no changes.

15th.—The child was discharged from the hospital, seemingly quite blind. The temperature has been normal since the 1st of June. She weighed 16¾ lbs. Medicinal treatment has consisted of the administration of mercury with chalk, in half-grain doses thrice a day, and later of cod-liver oil. The diet has included milk, whey, virol, cream, and orange juice. Fifteen-drop doses of brandy were given from May the 10th to June the 6th.

The child continued to attend the hospital as an out-patient. When discharged from the hospital, according to the mother's statement, she noticed nothing, and used to grope for things with her hands. She took no notice of a lamp or other bright light, and was considered to be altogether blind. As an out-patient she was ordered small doses of the compound syrup of phosphate of iron. On July the 4th—that is, nineteen days after she left the hospital and thirty-five days after she lost her sight—a note was made to the effect that "she begins to see." She commenced by noticing the presence of milk and other food, and the sight is said to have improved gradually until on August the 15th she could see just as well as other children. Martha F— was again seen by me on October the 9th, 1901, or about five months after her illness began. She was then a little over two years of age. She seemed to possess very good sight, and was said by her mother to be able to pick up pins from the floor. The child was in good general condition, of normal intelligence, and not deaf. The pupils were equal and responsive to light. When the fundi oculorum were examined a week later (October the 16th), the optic discs were found to be of good colour and the retinal vessels of normal calibre.

The foregoing cases of fleeting amaurosis must be carefully distinguished from a closely allied variety, where a child recovers from an attack of meningitis with impaired sight due to an incomplete atrophy of the optic papilla. There may, besides, be a paresis of one or more of the extrinsic muscles of the eyeball, generally, in my experience, of one of the muscles supplied by the third cranial nerve. My meaning may perhaps be rendered clearer if one or two illustrative cases be briefly quoted.

Case 5.—Albert H—, aged 7 years, was first seen on February the 21st, 1893. He was bad-tempered and feeble-minded, and his speech was difficult to understand. Slight nystagmus was present. The sight was obviously defective, but he was certainly able to count fingers at a distance of twenty feet. Both optic discs were incompletely atrophic. Upon inquiry, I ascertained that about three years before he came under my notice, he complained of headache, lost the power of walking, and developed a marked tendency to vomit; his head was held stiff, the left eye squinted inwards, and there was a discharge from the right ear. The last note I have about Albert H— refers to July the 18th, 1898, when sight in the right eye was equal to about one-half of the normal and in the left eye to about one-fifth of the normal. Fixation was imperfect, and the left eye tended to diverge. Colour-blindness was present. The optic discs were as noted previously. What I took to be a large hæmorrhage undergoing absorption was present in the central region of the left fundus oculi.

Case 6.—Arthur John M—, aged 1 year 6 months, was sent to me by my friend Mr. C. E. Adams, of West Norwood, on January the 18th, 1894. The history was that three months before coming under my notice the child fell ill, became drowsy, and did not care for his food. The condition gradually became worse; the child suffered from pain in the head, he was convulsed five or six times a day, and his neck was stiff and his head retracted. The right eye was noticed to turn towards the nose. There was no vomiting. The sight, which was good prior to the attack, seemed to fail in the third week of the illness. The patient was the sixth child, and was born at full term after a natural labour lasting twelve hours. The illness, diagnosed by Mr. Adams as meningitis, lasted about two months and a half.

When I examined the child the patient was found to be fairly well-nourished, with all his teeth except two. The anterior fontanelle was not widely open. The right eye had a large convergent squint, apparently due to a paralysis of the external rectus muscle. The pupils dilated readily under the influence of homatropine. The optic discs were sharply defined and somewhat white. The child retained just sight enough to move about without running into the furniture in the room.

Remarks.—Persistent and temporary blindness, then, occur in young children suffering from meningitis, the only difference being that while temporary blindness is not, symptomatic blindness is associated with permanent ophthalmoscopic changes. It seems more than probable that a single cause accounts for both forms. We know that the essential change of simple basal meningitis is an inflammation of the pia mater at the base of the brain, extending from the medulla behind to the optic chiasma in front, and due to the presence of a specific diplococcus which has been described by Still.* By matting together the cerebellar lobes the occlusive inflammation leads to closure of the foramen of Magendie, and so impedes the circulation of the cerebro-spinal fluid. The third ventricle thus becomes distended, and if the process continues may eventually press upon the optic chiasma. The pressure, if it last long enough, may readily set up a descending neuritis,† followed by atrophy, of the optic nerve and some degree of permanent blindness. This would account for the cases of slight but permanent amblyopia spoken of above.

If from any cause, however, the adhesions happened to give way early enough, the consequence would appear to be twofold: firstly, relief to the meningitic symptoms; and secondly, restoration of vision before the onset of atrophic changes in the optic nerve. This seems to be a reasonable view to account for the temporary amaurosis described in the present communication. It is scarcely conceivable that two processes so closely allied should not be due to a common cause, which cause I cannot but believe to be other than that of pressure by the distended third ventricle (infundibulum) upon the optic chiasma. The dilatation of the pupil, so often noted in cases of temporary amaurosis, is a further argument in favour of an organic origin for the blindness. The ocular nerves, the third, the fourth, and the sixth, especially the former two, may suffer either by the direct pressure of the distended ventricle, or by becoming involved in the inflammatory exudation at the base of the brain. This would, of course, account for the implication of the ocular muscles, as mentioned in Cases Nos. 3, 5, and 6 in the list. If the block in the circulation of the cerebro-spinal fluid be not relieved, blindness ensues, and the child eventually succumbs to the disease. Recurrence of obstruction indicates relapse of inflammation and also of amaurosis, as in Cases Nos. 2 and 3 just narrated. A similar train of reasoning would explain the amblyopia, persistent or temporary, as met with in cases of meningitis other than the simple basal variety.

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* Still, 'Journ. Path. and Bact.,' May, 1898.

† Optic neuritis is sometimes, although rarely, seen during the course of a simple basal meningitis.

(*Read June the* 21*st,* 1902.)

76. A CASE OF HYPERTROPHY OF THE GREAT TOE.

By R. C. DUN, C.M.

A girl, aged 7½ years, was brought to me a few days ago at the Liverpool Infirmary for Children, suffering from hypertrophy of the right great toe.

The mother gave as her history that up to the age of five years, *i. e.* two and a half years ago, both great toes were of the same size. Since then the right one had grown more rapidly than the left, more particularly during the last year. Six months ago a lump was noticed to form on the end of the toe, and the last joint began to turn up.

The four other children of the family are healthy and well-formed. There is no history of hereditary congenital deformities.

On looking at the right foot the great toe was seen to be slightly valgoid and markedly hypertrophied. The increase in size was noticeable both as regards the length and breadth of the digit. There was more than usual prominence about the head of the first metatarsal bone, and the tissues as far back as the middle of that bone showed increased development. The most marked deformity was of the terminal phalanx, which at first sight appeared to be markedly hypertrophied, the nail being vertical. But on further examination the plantar aspect of the toe was seen to be in apposition with the floor in almost its whole length when the child was standing, the appearance of hyper-extension being caused by a localised outgrowth from its dorsal aspect. Several well-marked sulci were present on the skin of the toe. The foot generally and the other toes were normal.

On handling the affected toe the skin was found to be thickened, and the subcutaneous tissues apparently were generally increased in amount, which gave a doughy sensation to the fingers; this was most marked on the plantar aspect.

On the dorsal surface of the terminal phalanx there was a definite tumour projecting upwards and displacing the nail; this mass was soft and elastic, adherent to the skin, and smooth on its surface. At the sides and tip of the toe it gradually merged with the hypertrophied subcutaneous tissue previously mentioned, without any line of demarcation. The overgrowth of soft parts was definitely palpable as far back as the centre of the first metatarsal bone, chiefly on its inner side.

In length the right great toe exceeded the left by half an inch. In circumference the right first phalanx was one and a quarter inches greater than that on the normal side, and the right second phalanx was one inch greater than the left.

The movements of the toe were normal. No pain was experienced. Sensation was unimpaired. There was slight awkwardness in walking, apparently due to the increased size of the affected toe.

I could distinguish no difference in temperature between the two great toes, such as has been mentioned as occurring in similar cases.

Dr. Holland kindly took an X-ray photograph of the feet for me, which shows well the increased development of the soft parts; it demonstrates also that the bones shared in the overgrowth, the increase in length of the bones on the right side being—

1st metatarsal	$\frac{3}{16}$ inch.
1st phalanx	$\frac{1}{16}$ "
2nd "	$\frac{2}{16}$ "

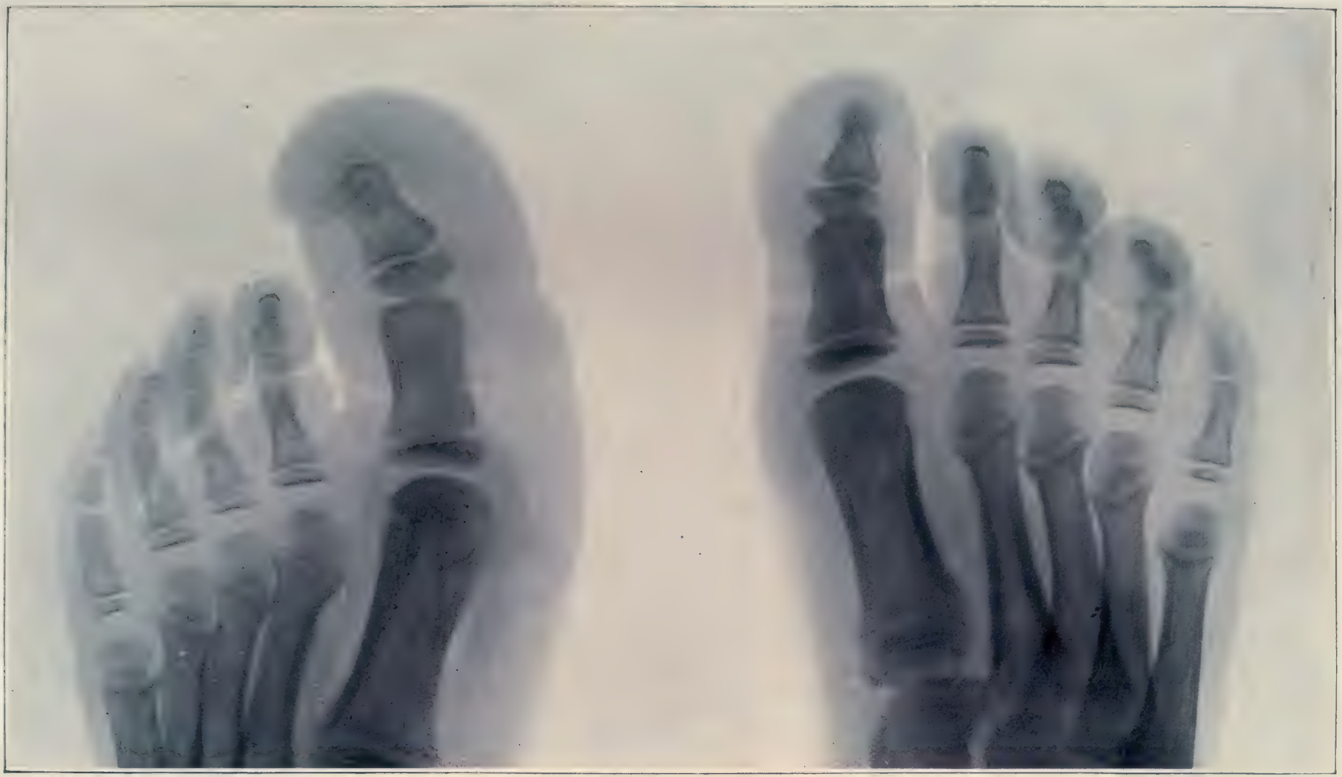
There is also very distinct increase in breadth of the first phalanx on the right side (*vide* Plate VIII).

I have photographs and a plaster cast showing the condition. I regret that I could not bring the patient.

The case appears to be one of general hypertrophy of all the tissues of the toe, the increased development being mainly lipomatous, particularly in the case of the terminal phalanx.

A somewhat curious feature of the case is that no enlargement of the toe appears to have occurred until the age of five years. In similar cases some increase in size has usually been present at birth.

I propose, if I can obtain the parents' consent, to amputate the toe either through the first phalanx or at the interphalangeal joint; this will equalise the length of the great toes and facilitate walking.



Mr. R. C. Dun's Case of Hypertrophy of the Great Toe.

Allard & Son, Imp.

I hope that I may be able to make a microscopical examination of the part removed.

(*Read June the* 21*st,* 1902.)

Discussion.

Mr. Clement Lucas asked whether there was the slightest sign of any injection of a nævoid character about the toe or foot. In many such cases of hypertrophy the condition was really one of nævoid lipoma, and if that were found in the present case it might not be necessary to excise so much as Mr. Dun at first suggested.

Mr. Sydney Stephenson said he hoped that if Mr. Dun obtained pathological specimens he would exhibit them before and report the results to the Society.

Mr. R. C. Dun (Liverpool), in reply, said he had carefully examined the toe for the nævoid condition mentioned by Mr. Lucas, but none was found. Indeed, the toe was somewhat paler than its fellow. He said he would be pleased to send any microscopical specimen and a report upon it to the Society.

Sequel by Mr. R. C. Dun (*August the* 7*th,* 1902).

Since reading the notes of the above case I obtained permission to operate.

I therefore, as a preliminary measure, removed the projecting mass of soft tissue from the dorsal surface of the terminal phalanx of the great toe.

On microscopical examination, it proved to be a pure lipoma, diffuse in nature, and blending gradually with generally hypertrophied subcutaneous tissue of the toe. There was no increased development of blood-vessels or of vascular spaces, indicating a nævoid condition.

A month later, as no diminution in the size of the toe had taken place, except as regards the circumference of the terminal phalanx, I amputated through the interphalangeal joint, by this means equalising the length of the great toes.

Under the microscope the skin was seen to be thickened, but normal in structure. The subcutaneous tissue showed marked increased development, the fat being particularly abundant. The thickened bone showed no departure from the normal. I could distinguish no pathological change in either the blood-vessels or the nerves.

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